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Original Articles.

A CASE OF PROGRESSIVELY DEVELOPING HEMIPLEGIA,
LATER BECOMING TRIPLEGIA, RESULTING FROM
PRIMARY DEGENERATION OF THE
PYRAMIDAL TRACTS.¹

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In this contribution the clinical and pathological details of a case of progressively developing hemiplegia, which later became triplegia, due to primary degeneration of the pyramidal tracts, will be presented, with a review of the literature of similar clinical cases. A summary of the literature of primary bilateral sclerosis will also be given.

J. S., white, was admitted to the Philadelphia Hospital August 9, 1897, where he died January 14, 1903. He was fifty-four years old when admitted. With the exception of a short time during which he was treated in the Men's Medical Ward for diarrhea or dysentery, he was during this period of six years a patient in the Men's Nervous Ward.

Omitting some unessential points, his history as traced in the hospital records is as follows: The family history obtained has

¹Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

From the William Pepper Clinical Laboratory, Phœbe A. Hearst Foundation.

no etiological value. He denied venereal disease. He was a heavy drinker and also used tobacco. He had had rheumatism six years before admission to the hospital. The earliest note referring to any nervous symptoms was made August 9, 1897, when a statement was recorded that two years before admission he had been paralyzed on the right side. It is probable that at this time (during 1890-1895) his loss of power first began to show itself.

On September 28, 1897, the record stated that right hemiplegia (more correctly hemiparesis) was present, that the biceps-jerk and knee-jerk were increased, and that the plantar reflex was absent. Ankle clonus also was absent.

On September 24, 1899, it was noted that he could use his right leg for various willed movements, but that it showed loss of power as compared with the left; also that there was some loss of power in the right arm as compared with the left. His grip measured by the dynamometer indicated fifty-five for the right and seventy-five for the left. Speech was a little thick. The knee-jerk on the right was much exaggerated, and the quadriceps-jerk and gastrocnemius-jerk were also increased. Both ankle-clonus and patellar clonus of moderate degree were present on the right. No note was made at this time as to the Babinski phenomenon. Biceps-jerk, triceps-jerk and all muscle jerks in the right upper extremity were increased. The tendon and muscle responses on the left were all present but not exaggerated as on the right, and patellar clonus and ankle clonus were absent on the left. Sensation for touch and pain was retained all over the body, and the stereognostic perception was undisturbed. The irides reacted to light and in accommodation, and other examinations for nervous phenomena gave negative results.

On May 17, 1901, the patient complained that the right leg was weaker than it had been one year previous. The reflexes and other symptoms were much as before recorded.

Although the case was seen from time to time no further notes as to his nervous symptoms were made until December 29, 1902, at which time it was recorded that the movements of the face and tongue were normal; the upper limbs could be moved voluntarily and fully in all parts; the grasp of the right hand was feeble, of the left normal; voluntary power in the right lower limb was very slight; in the left lower limb it was a little better than in the right but much impaired. The right upper limb and both lower limbs were spastic, but contractures and wasting were not present. The biceps, triceps and wrist reflexes were much exaggerated in the right upper extremity, and in the left they were a little more prompt than normal. Patellar and Achilles reflexes were exaggerated on both sides, but more so on the right; patellar clonus was present on the right but not on the left; ankle clonus was elicited on each side. The Babinski reflex was present but not

very prominent on the right; it was absent on the left. Sensation to touch and pain was everywhere normal.

This man suffered at times from severe intestinal and cardiovascular symptoms. On August 6, 1897, he began to have severe pain in the abdomen with diarrhea, with numerous stools accompanied by straining and pain. He was treated for these symptoms in the medical wards. The heart sounds at this time were feeble and accompanied by a low systolic murmur at the right base. The sounds were feeble on the left also, and were best at the ensiform cartilage. On December 11, 1898, the records show that he had a soft blowing systolic murmur at the second aortic interspace. May 31, 1899, the patient complained of pain in the chest most marked over the precordia. A soft double systolic murmur was heard above the second cartilage. The heart's action was weak and irregular. A few unimportant notes, chiefly negative, were made in September, 1899. The presence of the cardiac murmurs above described was reiterated. On January 17, 1902, the patient complained of constant and severe pain over the precordial region; the heart sounds were very feeble. The pulse on the left side was imperceptible, owing to the atheromatous condition of the arteries and to the cardiac weakness. The pulse on the right side was perceptible but weak, and the radial arteries on both sides were hard and rigid. The patient suffered at this time and later from cardiac weakness and anginal pains for which he was treated with various remedies.

About January 1, 1903, he began to show signs of great weakness and rapid failure. He had at times attacks of vomiting. He lost flesh, his pulse became weak and intermittent, and the right lung became edematous. He died January 14, 1903, his death being preceded by a slight convulsion.

The necropsy showed a serious pathological condition of the vessels and many of the viscera: chronic pleurisy and tuberculosis with cavity formation; endocarditis, contracted kidneys and chronic marked atheroma. The heart muscles were soft and pale, the valves distorted, thickened and calcareous, the aortic extensively atheromatous. Both kidneys were very firm, dark in color, with granular surface and irregular cortex. The stomach was decidedly contracted and the seat of chronic catarrh. Beginning at the junction of the transverse and descending aorta was a unilateral dilatation of this vessel, which increased in diameter to about 4 cm. This structure was atheromatous and contained a flattened clot. The brain and cord were removed for microscopical examination, of which the following is a record.

The degeneration in the lumbar region was very distinct by the Weigert hematoxylin stain in the crossed pyramidal tracts, and more intense in the right than in the left. The direct pyramidal tracts by this stain did not appear degenerated at this level of

the cord. The nerve cells of the anterior horns were not distinctly altered, they were numerous and some were much pigmented, which, considering the man's age, was not remarkable. The chromophilic elements were well formed and the nuclei central. It may be said that the nerve cells of the anterior horns of the lumbar region had the usual appearance in persons of the same age. The anterior roots on each side in the lumbar region were normal.

At the eighth cervical segment the degeneration of the right crossed and left direct pyramidal tracts was very distinct by the Weigert hematoxylin stain. The left crossed and right direct pyramidal tracts were much less degenerated. The nerve cells of the anterior horns of this region may be said to have been normal, although in examining a number of sections a few cells were found that were not normal; the nerve cells in general were remarkable for their excellent state of preservation.

Sections from the lower cervical region stained by the Marchi method showed no recent degeneration in the right crossed and left



Long-standing degeneration of the left direct pyramidal tract, recent degeneration of the right direct and of the left crossed pyramidal tracts. The dots in the crossed tract are not so large as those in the direct tract (Method of Marchi).

direct pyramidal tracts. The left crossed and the right direct pyramidal tracts contained numerous small black dots, and those of the crossed tract were much smaller and less numerous than those of the direct tract. At the outer border of the right direct

pyramidal tract the dots were smaller than in the median and inner portions of this tract.

The anterior roots in the lower cervical region were normal. There was no distinct meningitis; here and there a slight accumulation of round cells was found in the pia.

Both pyramidal tracts were much degenerated in the oblongata and pons, but the left tract was much more degenerated than the right. The right pyramidal tract was much degenerated in the oblongata and pons by the Marchi method, while the left showed no recent degeneration by this method. About the middle of the pons, the left pyramidal tract contained a large proportion of normal nerve fibers, but a part of it was much degenerated. No degeneration could be detected in the foot of the right or of the left cerebral peduncle by the Weigert hematoxylin method, but degeneration by the Marchi method could be found in the middle portion of the foot of the right cerebral peduncle and also where the foot of the right cerebral peduncle becomes the lower part of the posterior limb of the internal capsule; the degeneration however was slight. No degeneration could be seen by the Marchi method in the foot of the left cerebral peduncle.

Sections of the left paracentral lobule contained Betz cells that appeared normal. They were not numerous, but this does not mean that there had been a loss of the cells, as the number of these cells seems to vary greatly in different cases.

Cellular infiltration of moderate intensity was found in the pia about the cerebral peduncles and pons, but it seems hardly probable that this could explain the long-standing degeneration of the pyramidal tract from the left cerebral hemisphere, and for this reason we have spoken of the degeneration of the pyramidal tracts as primary. No focal lesion could be detected anywhere.

Summarized, this case was one in which hemiplegia gradually developed on the right side, the lower extremity being more markedly and probably earlier affected than the upper, the case therefore at first belonging to the clinical type of unilateral progressive ascending paralysis. After several years the left lower extremity also became paralyzed, but not to the same extent as the right. The reflexes were all markedly exaggerated, the Babinski response being present. Sensory symptoms were absent. Microscopical examination showed intense and long-standing degeneration of the right crossed and the left direct pyramidal tracts, the degeneration extending into the pons but not into the left cerebral peduncle; also comparatively recent degeneration of the left crossed and the right direct pyramidal tracts, traced by the

method of Marchi into the lower part of the right internal capsule. No lesions, degenerative or focal, were found elsewhere in the brain or spinal cord; the case, therefore, was one of primary degeneration of the motor tracts, much greater and older in the right crossed and left direct pyramidal tracts. The case may be regarded as a corroboration of the clinical type described by Mills in the *JOURNAL OF NERVOUS AND MENTAL DISEASE* for April, 1900.

In the *JOURNAL OF NERVOUS AND MENTAL DISEASE* for April, 1900, in a paper entitled "A Case of Unilateral Progressive Ascending Paralysis, Probably Representing a New Form of Degenerative Disease," which was presented to the Philadelphia Neurological Society on December 18, 1899, an interesting case is detailed, reference being made to another somewhat similar case previously observed. The patient, a man fifty-two years old, about two years before coming under observation began to show signs of weakness in the right lower extremity, as indicated by his method of walking. The paresis came on slowly and insidiously. Weakness in the arm appeared for the first time eighteen months after the weakness was noticed in the lower extremity. This weakness soon became more and more evident, and was accompanied by the tendency to carry the arm raised against the body and flexed at the elbow. When first examined the paresis in the upper extremity, although easily determined, had not nearly reached the degree of impairment observed in the leg. The right side of the face was also slightly but undoubtedly paretic. The patient had had at one period some hyperesthesia in the right lumbar region and in the lower extremity, and later in the right upper extremity. Herpes appeared in the lumbar and lumbosacral regions, lasting for a short time. Wasting was distinct in the right lower extremity, the measurements showing a difference of one and a half inches for the thigh, and five eighths of an inch for the calves. The various movements of the right leg were distinctly weaker than those of the left, but were nowhere absolutely abolished. Similarly all the movements of the right arm were distinctly impaired, but were nowhere absolutely lost. The dynamometer showed 180 for the right and 160 for the left. Faradic contractility was retained. The affected limbs were not spastic nor contracted. Careful examination

showed retention of all forms of sensation. The tendon and muscle phenomena on the right side were all somewhat exaggerated. Knee-jerk was plus on the left side, but was considerably more exaggerated on the right. Patellar clonus was present on the right but not on the left, and the right side showed a slight ankle clonus, which was absent on the left. The plantar reflex was normal on the left; but on the right, while the Babinski reflex was not present, the normal response was distinctly less marked than on the left. It might be described as between normal plantar flexion of the toes and the dorsal flexion of the Babinski reflex.

This case has recently been made the subject of reinvestigation by Dr. Hugh T. Patrick, who inclines to the opinion that the case is one of paralysis agitans without tremor, the disease up to the present time having attacked only one side of the body. His report of the case was presented at the meeting of the American Neurological Association, and will be found in its Proceedings, with the discussion of the case by Dr. Mills. At the time when the patient was examined by Dr. Mills, among the reasons for believing that the case was not paralysis agitans were the absence of spontaneous tremor; the presence of markedly exaggerated reflexes on the affected side, even including ankle clonus and patellar clonus; the existence of decided wasting on one side, especially of the lower extremity, and the absence of the facies of paralysis agitans and of a fixed position of the body and head.

According to Sir William Gowers, both primary lateral sclerosis and paralysis agitans are probably abiotrophies (*Lancet*, April 12, 1902), and it may be that in the case under consideration the two abiotic diseases are conjoined.

In the same paper in which this case is detailed a second case is reported. This patient was under the care of Dr. Mills seventeen or eighteen years ago, and had previously been a patient of the late Dr. E. C. Seguin of New York, who believed that there was a cerebral lesion causing changes in the crossed pyramidal fasciculus analogous to lateral sclerosis. The view taken by Mills was that the changes in the crossed pyramidal tract were of the nature of a lateral sclerosis, but were primary and not secondary to any cerebral lesion. The patient was a woman forty-

three years old, who first noticed weakness in her leg while pregnant with her last child, three years before coming under observation. The left arm became paretic a few months after the left leg. The reflexes were much increased on the affected side. The patient remained under observation for many months, the paresis of the leg and arm slowly increasing. Neither arm nor leg was contractured, and sensibility was preserved. The patient complained at times of pains like neuralgias in the limbs, and of nervous twitching in the leg and arm. This patient was alive three or four years ago, and had become entirely unable to walk, but just how she was affected was not learned.

In the paper reporting these cases the differential diagnosis from an unusual form of unilateral disseminated sclerosis, from unilateral amyotrophic sclerosis, from a progressive hemiplegia due to slowly increasing focal cerebral lesion involving the motor subcortex or the internal capsule, from a degenerative motor neuritis, and from a functional hemiparesis was discussed. Reasons were given why the cases did not seem to fit in exactly with any one of these diagnoses.

In addition, as has been indicated above, the diagnosis must be made from unilateral paralysis agitans. Syphilitic hemiplegia later becoming a triplegia might also have some points in common, but could usually be separated by the presence of such concomitant syphilitic affections as paralyses of the cranial nerves.

In the *Philadelphia Medical Journal*, Feb. 9, 1901, Spiller has recorded the case of a man, forty-one years old, who began, four years before coming under observation, to feel weak in the left lower limb while walking. The weakness increased, and in about a year the left upper extremity also showed signs of feebleness. He was never unconscious and never had headache or vertigo or other signs of focal intracranial disease. When the case was recorded the movements of the left lower limb were spastic, but not ataxic; the toes of the left foot were scraped along the ground and the foot turned inward. Both knee-jerks were prominent, but the left much more so than the right. Ankle clonus was obtained on the left but not on the right, and the Babinski reflex was distinct on the left, but uncertain on the right. Sensation was normal. The resistance to passive movement and the grasp of the hand were decidedly weak. The left upper limb

was weaker than the right; it was also spastic, but the weakness and spasticity were less than in the lower extremity. The left upper limb was held slightly flexed at the elbow and against the body, but no contractures were present anywhere. Speech was normal, but the mouth could not be drawn up as well on the left side as on the right. The tongue went slightly to the left on protrusion. Ophthalmoscopic examination by Dr. H. F. Hansell showed white atrophy of the left optic nerve. After giving the details of his case, Spiller discusses its probable diagnosis, and concludes that the cases reported by Mills and by him have the symptoms which one should expect from unilateral lateral sclerosis, but he also called attention to the fact that other lesions might cause the same symptoms. The wasting is like that occurring in hemiplegia.

Potts (*JOURNAL OF NERVOUS AND MENTAL DISEASE*, Oct. 1901), under the title, "A Case of Progressive Unilateral Ascending Paralysis Probably Due to Multiple Sclerosis," has reported a case somewhat similar to the case described by the authors of this paper. The patient, a youth of nineteen, when about fifteen years old began to drag his right foot, this foot in walking showing a tendency to cross over the other. Two years after the impairment of power, diplopia appeared and remained for five months. It was discovered at about this time that the right leg was smaller than the left. Weakness in the right arm was first noticed about three and a half years after its appearance in the right leg. Examination showed that when the patient walked the right leg was rigid and the toe dragged. All movements of the leg were preserved but impaired. The right arm was distinctly weaker than the left, but was not spastic. Both the arm and leg were decidedly atrophied, as shown by careful measurements. The adductor pollicis was not so large or firm as the left. The reflexes were prompt everywhere but exaggerated on the right, the Babinski response being present. The right side of the face showed slight paresis with some tremor on voluntary effort. Paresis of the muscles of the right side of the throat was present. Nystagmus was present especially when the eyes were turned to the right. There was paresis of the left inferior rectus, and paleness of the temporal halves of the discs. All forms of sensation, including stereognosis, were normal. A slight Romberg symp-

tom and tendency to incoördination were sometimes shown. The right hand and foot were colder to the touch than the left. Potts, while recognizing the resemblance between his case and the cases reported by the writers, holds that his case is probably one of disseminated sclerosis, and suggests that our cases might also be classed in this category, a view which we cannot accept. This diagnosis in Pott's case may be correct, although it is also probable in this and similar cases of disseminated sclerosis that the degeneration first and for a long time is chiefly confined to the pyramidal tract on one side.

Gowers, in his paper on "Abiotrophy" (*Lancet*, April 12, 1902), says truly "that the symptoms of systematic degeneration are not alone ground for the diagnosis of insular sclerosis, nor is the additional presence of nystagmus. Nystagmus on movement of the eyes is met with in many spinal degenerations of pure systemic type."

In his paper on "Chronic Progressive Hemiplegia," read at the present meeting of the American Neurological Association, Patrick describes a case which he regards as clearly indicating the progressively developing hemiplegia due to degeneration of the pyramidal tract of one side. During a recent visit to Chicago one of us (Dr. Mills) had the opportunity, through the courtesy of Dr. Patrick, of seeing this case, that of a young woman, eighteen years old, whose motor disability appeared about four years previously. The essential features of this case are the unilateral and progressive character of the paralysis which probably began first in the lower extremity, the exaggeration of the reflexes on the affected side, the absence of all forms of sensory disorder and of visceral symptoms, and the moderate wasting of the limbs and face. The reader is referred to Dr. Patrick's paper for fuller particulars of this case.

Another case of unilateral progressive ascending paralysis has been in our care at the University Hospital.

From a study of the cases presented in this paper, and in the light of the pathological findings in the case with autopsy, it may be concluded that there is a form of progressively developing hemiplegia, usually of ascending type, sometimes becoming triplegia or even quadriplegia, due to a progressive primary degeneration of the pyramidal tracts, which begins on one side and

may extend to the other. This clinical picture may be produced by other lesions, but we believe that we have established the fact that primary progressive degeneration of one pyramidal tract occasionally occurs. This is a unilateral lateral sclerosis, and in this connection it is interesting to devote some space to the subject of bilateral lateral sclerosis.

In a recent address in London, Erb² spoke of the spastic spinal paralysis first described by him in 1875. He believes that sufficient cases have been published to show that primary bilateral sclerosis exists. The following cases he gives as indisputable, and capable of standing any criticism.

Case 1. Minkowski.³ Clinically the picture was one of spastic spinal paralysis. Anatomically there was a nearly pure sclerosis of the crossed pyramidal tracts, with slight changes in the direct cerebellar tracts.

Case 2. Von Strümpell.⁴ The patient was a man, aged sixty-three years, who had a brother suffering from the same disease. Clinically the case presented the typical picture of spastic spinal paralysis for at least twenty years. Anatomically there was a typical degeneration of the pyramidal tracts from the lumbar to the cervical region, and in addition a slight degeneration of the direct cerebellar tracts and a still more trifling degeneration of the tracts of Goll in the upper part of the spinal cord. Strümpell would, on this account, reckon the affection among the combined system diseases, but the degeneration of the pyramidal tracts was certainly the most essential lesion, and is to be regarded as primary.

Case 3. Dejerine and Sottas.⁵ Clinically this was a pure case of spastic spinal paralysis of twenty-five years' duration, death occurring from pneumonia in the sixty-sixth year. Anatomically there was marked sclerosis of the pyramidal tracts from the lumbar to the cervical cord, with slight partial sclerosis of Goll's tracts in the cervical region.

Case 4. Donaggio.⁶ The patient was a man aged sixty-one years. Clinically there was for two and a quarter years the typical picture of pure spastic spinal paralysis; death resulted from pneumonia. Anatomically there was pure, exclusively primary degeneration of the pyramidal tracts from the lumbar to the cervical region (a quite typical case).

Case 5. Friedmann.⁷ The patient was a male, aged fifty-two years. Clinically for two years the picture was one of a spastic spinal paralysis (trace of disturbed sensation being present?). The patient suffered from apoplexy, death resulting from pneumonia. Anatomically there was classical primary degeneration of the pyramidal tracts only, there being a trace of degeneration in the direct cerebellar tracts as well as endarteritis obliterans of the basilar artery.

Case 6. Von Strümpel.³ Clinically a picture of spastic spinal paralysis was present, death occurring after the disease had lasted for thirty-five years. Anatomically there was quite typical moderate degeneration of the pyramidal tracts from the lumbar cord to the region of the pyramids. The direct cerebellar tracts were scarcely affected and the tracts of Goll very slightly in the upper cervical region. The anterior columns and brain were entirely free (belongs to the hereditary form).

Cases 7 and 8. Bischoff.⁹ The patients were two brothers who, from their eighth and tenth years respectively, suffered from spastic rigidity of the limbs, ascending slowly from the legs to the head. Intellectually development was poor, but otherwise clinically the typical picture was one of spastic spinal paralysis. Death took place from phthisis after about twenty years' duration. Anatomically there was a typical degeneration of the pyramidal tracts, extending upwards beyond the oblongata, but not farther. The direct cerebellar tracts and Gowers' bundles were all but free. Goll's tracts were extremely affected. In the gray matter of the anterior columns there was atrophy of the ganglion cells (evidently occurring towards the end of life). The author calls the change in the spinal cord primary tract degeneration (quite analogous to the condition found by Strümpel), and holds that thereby the existence of an infantile form of hereditary spastic spinal paralysis has been proved.

Case 9. Ida Democh.¹⁰ The case is clinically and anatomically somewhat complicated; clinically there was a typical picture of spastic spinal paralysis, combined with signs of chronic alcoholism, pains, tremors, etc. Anatomically there were primary degeneration of the tracts of Goll and congenital hydromyelia in the lumbar and dorsal cord; the direct cerebellar tracts were free; nevertheless, as proved by the author, this can be termed a case

of primary degeneration of the pyramidal tracts in spastic spinal paralysis.

Case 10. A case clinically typical of spastic spinal paralysis described by Kühn, in which von Strümpell made the microscopical examination of the spinal cord. He found an essentially characteristic primary degeneration of the lateral columns.

Erb regarded the sclerosis of the direct cerebellar tracts and the slight degeneration of the columns of Goll in some of these cases as of secondary importance.

He thinks that the case described by Strümpell¹¹ in 1894 as a primary isolated system degeneration of both pyramidal tracts, showing only slight transitions to amyotrophic lateral sclerosis, might come under this head.

To this record we would add the case reported by us in this paper, and also the case reported by one of us (Spiller¹²) in which there was primary degeneration of the pyramidal tracts with comparatively slight alteration of the nerve cells of the anterior horns of the spinal cord.

Note—Since this paper was read, another communication by Erb on spastic spinal paralysis has been published in the *Deutsche Zeitschrift für Nervenheilkunde*, Vol. 23, Nos. 5 and 6. He refers to the ten cases mentioned in his previous paper, and includes the well known case of Morgan and Dreschfeld.

²Erb, *Lancet*, 1902, p. 970.

³Minkowski, *Deutsches Archiv f. klin. Med.*, Vol. 34, 1884.

⁴Von Strümpell, *Archiv f. Psych.*, 17, 1886.

⁵Dejerine and Sottas, *Archives de Physiologie Normale et Pathologique*, 1896, p. 630.

⁶Donaggio, *Rivista sperimentale di freniatria*, Vol. 23, 1897.

⁷Friedmann, *Deutsche Zeitschr. f. Nervenheilk.*, Vol. 16, 1899.

⁸Von Strümpell, *Neur. Centralbl.*, 1901, p. 530.

⁹Bischoff, *Jahrbücher f. Psych. und Neur.*, Vol. 22, 1902.

¹⁰Ida Democh, *Archiv. f. Psych.*, Vol. 33, 1900.

¹¹Von Strümpell, *Deut. Zeitschr. f. Nerv.*, Vol. 5, p. 225.

¹²Spiller, *JOURNAL OF NERVOUS AND MENTAL DISEASE*, May, 1902.

THE RESULTS OF SURGICAL TREATMENT OF BRAIN TUMORS.

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In order to estimate accurately the prospect of success in any surgical operation, it is wise to consider the results which have been attained in a large number of cases. And while, too great importance should never be attached to statistics, yet on the other hand, they possess a certain value and are never without an element of interest. In 1893, I was able to collect seventy cases of brain tumor in which an operation had been undertaken,¹ and in 1896 and 1897, I analyzed the results in two hundred and twenty cases in which a surgical operation had been attempted.² As a knowledge of the possibility of operation has become more widespread, and as the technic of brain surgery has become more perfect, reports of such operations have been more frequent, and it has been possible to collect 365 cases up to January 1, 1903.³ In the appendix to this article the cases which have been published since January 1, 1896, are tabulated, all those prior to that time being referred to either in *Brain Surgery*, or in the *Medical Record* for February 1, 1896, or in the article in the *Montreal Medical Journal* for November, 1897.

TABLE OF RESULTS OF OPERATION FOR BRAIN TUMOR.

		Cere- bral.	Cere- bellar.
Total number of cases operated upon.....	365	315	50
Cases in which the tumor was not found.....	111	91	20
Cases in which the tumor was found but not removed.	27	21	6
Cases in which the tumor was removed and the patient died	59	51	8
Cases in which the tumor was removed and the patient recovered	168	152	16

There are two elements which render the chances of success in the operation for brain tumors better at present than formerly.

(1) The accuracy of diagnosis and the accuracy of localization

¹Brain Surgery, Wm. Wood & Co., 1893.

²New York Medical Record, February, 1896. Montreal Medical Journal, November, 1897.

³These cases have been gathered from the literature for me by Dr. O. Hensel.

of tumor are today more easily possible than before. Given a history of a slowly progressing disease with symptoms of headache, vomiting, vertigo, sensations of cerebral discomfort, progressive emaciation and feebleness, and a gradual loss of sight due to a choked disc, and the only probable hypothesis is the existence of a brain tumor. No other disease can produce this particular combination of symptoms. Hence the diagnosis of the nature of the disease is not difficult.

The possibility of locating tumors has also become more definite within the last twenty years. This diagnosis is wholly by direct observation of local symptoms such as (a) mental symptoms, (b) motor or sensory aphasia, (c) local spasm and monoplegia, (d) hemianopsia, (e) cerebellar staggering.

The diagnosis is also one to be reached in part by elimination, for the absence of any one of these localizing symptoms just named, when the disease is clearly a brain tumor, makes it evident that the tumor is inaccessible. It is therefore possible at the present time to determine with considerable accuracy, *first*, that a tumor is present, and, *second*, that the surgeon can reach it.

The chance of success has also been materially increased by the newer surgical methods of access to the brain. It is true that successes had been obtained by the use of the ordinary trephine, the opening being enlarged by a rongeur. It is also true that the method of opening the skull by a horseshoe-shaped incision through scalp, periosteum and bone, by means of a gouge and chisel, has been attended by greater success. This method was a distinct improvement upon the method by the use of the trephine. But during the past three years these two methods have been superseded by the use of the electrical saws. In the larger hospitals it is perfectly possible to obtain a powerful electrical engine connected with a flexible tube, on the end of which revolving spools can be inserted. The method at present used by McCosh at the Presbyterian Hospital seems to me the best method as yet devised for entering the skull. A small bulb of the shape of a pear, about one-quarter of an inch in diameter, is attached to the electrical engine, and with this bulb four or five small holes are bored through the skull. The cone-shape of the bulb allows the point to penetrate through the skull as it is revolved, and yet prevents the shaft from entering the skull or penetrating the dura. It requires but a few seconds with the powerful revolutions of this bulb, that are ob-

tained by the machine, to bore one of these holes. The number of holes and their position upon the skull are determined entirely by the area that it is desired to expose. If four holes are bored they are from one and a half to four inches apart. When these holes have been made the bulb is removed from the machine and there is substituted a small circular saw and the bone between these holes is quickly sawed through by it. A small projecting and protecting flange is screwed to the side of the circular saw so that the blade of the saw is exposed only for the depth of the skull as measured through the holes, and in this way the danger of sawing through the dura is wholly obviated. It requires hardly a minute when the saw is properly adjusted to saw through the skull from one hole to the next. In this manner the skull can be opened in from six to eight minutes after the original incision has been made through the scalp. This method offers great advantages in the rapidity with which access can be had to the brain.

Experience has taught that it is a great advantage in the extraction of tumors to have plenty of space, and it has also taught that these large bony flaps can be replaced and will unite without any danger. The bony flap is turned downward, as a rule, the lower edge of the flap not being sawn entirely through, but cracked. It is left attached to the periosteum and skin. The incision in the scalp will be determined in its shape by the incision which it is proposed to make in the bone. This may be rectangular or polygonal. Much time is saved in the operation by making the bony flap of large size, as in former operations it was often necessary to delay for the enlargement of the bony opening after the tumor had been found. By the use of this method, I have frequently seen the brain exposed within ten minutes of the time of the beginning of the operation; while in prior methods from eighteen to twenty-five minutes were often expended in this preliminary stage.

Another factor in success in these operations is the fact that we have come to the conclusion that extensive cutting of the brain and exsection of parts of the brain are not necessarily attended by danger, provided the hemorrhage can be arrested. I have seen large areas, two to three inches in diameter, cut out of the brain, to a depth of two to three inches, and I have seen the patients recover. While the difficulties attendant upon the extraction of a deep tumor are many, and while the chances of subsequent recovery, first, from the operation, and, second, from the symptoms, are

much less in such deep tumors, yet it must be conceded that a tumor lying one or two inches below the surface may be reached.

For these reasons it seems to me probable that the statistics of the next ten years will show a very much greater percentage of successes than do the statistics up to the present time.

The operation for cerebellar tumors appears to me to be of great difficulty and danger. But one-tenth of each hemisphere of the cerebellum can be reached by the surgeon through the occipital fossa. This I have determined by measurements of the surface many times in the cadaver. The surface which is accessible is one which is very rarely the seat of a cerebellar tumor. Cerebellar tumors lie either in the middle lobe of the cerebellum directly beneath the tentorium, a point which cannot be reached from the lower surface of the cerebellum, or else they lie in the sulci between the cerebellum and the pons and medulla oblongata, upon the base of the brain, a point which is equally inaccessible. For this reason failures are numerous in operations for cerebellar tumor, and after a considerable experience (I have had nine such cases operated upon without any success) it seems to me futile to attempt this operation.

The causes of failure are: First, mistakes in the diagnosis of the location of the tumor. In a certain percentage of tumors these mistakes are inevitable. Certain local symptoms of tumor which appear to point to a clear localization do occur in some cases when the tumor is at a considerable distance from the point where it is supposed to lie. The local symptoms are therefore not infallible in their indication.

Secondly, some tumors, even when accessible, cannot be removed. They are infiltrating gliomata without any boundary and with a vascular, softened tissue about them which prevents enucleation, and this variety of tumor is particularly liable to return even if removed in part.

Thirdly, the dangers of hemorrhage and the dangers of meningitis are less dreaded at present than formerly, proper asepsis preventing the latter complication in all cases.

APPENDIX BY DR. O. HENSEL.

VARIETY OF TUMOR REMOVED AND RESULTS.

1893-1903.

	Recovered.	Died.
Sarcoma	34	13
Melanosarcoma	1	—
Cystosarcoma	1	—
Gliosarcoma	5	—
Glioma	8	5
Neuroglioma	2	—
Perithelioma	1	—
Endothelioma	3	3
Carcinoma	1	—
Hydatid Cyst.	4	1
Cyst	22	7
Tubercle	11	4
Gumma	5	2
Fibroma	1	2
Angioma	4	—
Osteoma	1	—
Not stated	16	9
	120	46

TUMOR TREPHINED FOR BUT NOT FOUND.

- Central Region.*—1-2. Bramwell, *Boston Med. Journ.*, Dec., 22, 1900, 2 cases, glioma right motor region, no tumor found, death in 9 months.—3. Hoppe, *Journ. of Amer. Med. Assoc.*, Feb. 2, 1901, Tubercle in lentiform nucleus of right side, extending to within $\frac{1}{2}$ inch of cortex in ascending frontal and parietal regions and inward and downward to internal capsule. Death in 6 hours.—4. Hoppe, *ibid.*, Gliosarcoma, extending 2 inches from tip of 1st frontal convolution, back along longitudinal fissure to 2 inches posterior to fissure of Rolando. Death on same day.—5. Hoppe, *ibid.*, Multiple cysts. Trephined at lower extremity of fissure of Rolando. Death from cerebral hemorrhage.—6. Schulze, *Deutsche Zeitschrift f. Nervenheilk.*, 1896. Sarcoma in left facial center, death.—7. Clarke, *Glasgow Med. Journ.*, 48, p. 13. Motor area trephined, not found, death, no autopsy.
- Frontal Region.*—8. Hitzig, *Therap. Wochenschrift*, 1896. Glioma, right frontal lobe, death soon after operation.—9. Zenner, *Transactions of American Neurological Association*, May, 1897. Subcortical growth in prerolandic gyrus.—10. Stochow, *Inaugural Dissert.*, Erlangen, 1896. Tumor, left frontal lobe on autopsy.—11. Hahn and Ruheman, *Deutsche med. Wochenschrift*, 1896. Trephined in frontal region, not found. Was in 2d and 3d frontal convolution. Improvement.—12. Clarke, *British Med. Journ.*, Oct. 13, 1900. Glioma, left frontal lobe, not found, death next day.—13. Armitage, *Lancet*, Nov. 26, 1898. Cortical growth, superior and middle frontal convolution.—14. Gordinier, *American Journ. of Med. Sciences*, May, 1899. Glioma, second left frontal convolution.
- Parietal Region.*—15. Williamson, *British Med. Journ.*, July 6, 1901. Sarcoma at right parieto-occipital fissure, death in 1 month.—16. Ros-solimo, *Arch. f. Psych.*, xxix. Cavernous angioma of left paracent. lobe, death in 15 hours of hemorrhage.—17. Lambotte, *Annals de la Soc. Belg. de Chir.*, 1896. Trephined in occipital region. Was in angular gyrus. Death.
- Temporal Region.*—18. Gessler and Bayha, *Würtemb. ärzt. Correspbl.*, 1897. Tumor in temporal lobe, death in 6 days.—19. Starr and Weir,

- Medical News*, Aug. 7, 1897. Glioma.—20. *Transactions of the American Neurological Association*, May 5, 1897. Sarcoma in left temporal lobe.—21. Starr, *British Med. Journ.*, Oct. 16, 1897. Glioma, trephined for in left 3d frontal convolution, death.
- Occipital Lobe*.—22. Barette, *Soc. de Chirurg. de Paris*, Nov. 11, 1897. Neuroglioma of occipital lobe, compressing longitudinal sinus and causing a swelling in the frontal region, which could not be removed on account of hemorrhage.
- Deeper Parts of Brain*.—23. Mingazzini, *Deutsche Zeitschrift f. Nervenheilk.*, 1900. Sarcoma in right island and corpus striatum.—24. Broca, *Arch. gen. de Méd.*, 1896. Tumor of chiasma, could not be found, ventricles drained, death in 2 weeks.—25. McBurney, *Med. Record*, 1896. Sarcoma of left centrum ovale and base. Death.—26. McCosh, *Med. Record*, 1896. Tumor of corpora quadrigemina. No change after operation.—27. Bury, *British Med. Journ.*, Nov., 1896. Tubercle of optic thalamus. Death in 2 months.—28. Glaser, *Langenbeck's Arch.*, i, 1901. Glioma, region of calamus scriptorius, death in $\frac{3}{4}$ hours.—29. Rohner, *Rev. Méd. de l'Est.*, xxx, 1898. Basal tumor.—30. D'Alloco, *La rif. med.*, 1897. Optic thalamus.—31. Gordinier, *Alb. Med. Annals*, 1898. Centrum ovale, died.—32. Thomas and Hamilton, *Journ. of Exp. Med.*, 1897. Corpus callosum, died.—33. Taylor, *Journ. of Exp. Med.*, ii, 6. Optic thalamus, died.—34. Thompson and Ferguson, *Glasgow Med. Journ.*, 56, p. 424-429. Tubercle, suspected in motor area, found on autopsy in lenticular and caudate nuclei.—35. Starr, *British Med. Journ.*, Oct. 16, 1897. Large sarcoma in basal ganglia, under area exposed. Death, 2 weeks.—36. Schulze, *Deutsche Zeitschrift f. Nervenheilk.*, 1896. Gliosarcoma at right side of base with hydrocephalus of left lateral ventricle.
- Cerebellum*.—37. Ziegenweide, *Psych. en neurol. Bl.*, 1899. Suspected in cerebellum, on autopsy found between right cerebellum, tentorium and pons.—38. Heidenhain, *Arch. f. klin. Chirurgie*, 64, p. 848. Sarcoma of upper and lower worm. Death in 7 hours.—39. Stieglitz, Gerster and Lilienthal, *American Journ. of the Med. Sciences*, Vol. iii, p. 509. Gliosarcoma of right cerebellum, meningitis.—40. Borelius, *Ncur. Centralbl.*, 1897. Fibroma of right cerebellum. Death soon after.—41-42. Janz, *Schmidt's Jahrb.*, 1897. Two cases, tumor cerebelli, not found, death.—43. Gerster, *American Journ. of the Med. Sciences*, May, 1896. Cerebellar tumor, not found.—44. D'Alloco, *Rif. med.*, 1897.—45. Starr, *British Med. Journ.*, Oct. 16, 1897. Glioma, right cerebellum, ventricle drained, improvement, death in 8 days.—46. Starr, *ibid.*, Cerebellar tumor, not found. Operation had to be stopped owing to hemorrhage. Discharged in same condition.—47. Descos, *Lyon Méd.*, xcii. Not found, death.
- Region Not Stated or Indefinite*.—48. Koster and Schiller, *Neurol. Centralblatt*, 1897. Sarcoma, left hemisphere. Death, 2 months after slight improvement.—49. Gould, *Clin. Soc. of London*, Jan. 11, 1898.—50. Sidney, *British Med. Journ.*, 1898, p. 215.—51. Swolfs, *Journal de Neurologie*, 1897, No. 24.—52. Hirsche, *Wiener klin. Wochenschrift*, 1896, No. 5.—53. Nicoll, *Glasgow Med. and Chirurg. Soc.*, March 4, 1898.—54. Fisher, *Transactions of the American Neurological Association*, May, 1897.—55. Langdon, *ibid.*—56. Spencer, *Therap. Gazette*, 1898. Operated in occipital region, recovered.—57. Phillips, *British Med. Journ.*, 1898. Recovered.—58. Spencer and Keene, *Therap. Gazette*, Recovered.—59. Sinkler, *Journ. of Nervous and Mental Disease*, xxi, p. 796. Death in 54 days.—60. Hitzig, *Therap. Wochenschrift*, 1896. Sarcoma, left hemisphere, death in 3 weeks.—61. Hitzig, *ibid.* Patient's condition not changed.—62. Ruheman and Hahn, *Deutsche med. Wochenschrift*, 1896. Operated on cerebellum, position (?) No autopsy.—63. Gibson, *Edinb. Med. Journ.*, Feb., 1896.

TUMORS FOUND, NOT REMOVED.

- Central Region.*—64. Krauss, *New York Med. Journ.*, July 30, 1898. Trephined over left arm center and found hard mass 1 inch below cortex which could not be removed. Recovery.—65. Wiener, *New York Med. Journ.*, 1898. Glioma of arm, face and speech centers, too large for removal.—66. Elliot, *Boston Med. and Surg. Journ.*, 1896. Multiple sarcomata of left motor zone, removal intended for a second operation, but death before this after slight improvement.—67. McBurney, *Med. Record*, 1896. Glioma of central convolutions which could not be extirpated. Result (?).—68. Henschen, *Mitth. aus dem Grenzgebiet der Med. und Chir.*, 1898. Too large and diffuse to be removed.—69. Henschen, *ibid.*, too large and diffuse to be removed; death in 8 months.—70. Starr, *British Med. Journ.*, Oct. 16, 1897. Glioma, motor area, could not be removed owing to hemorrhage. Death 24 hrs.—71. Eskridge, *Denver Med. Times*, June, 1896. Glioma, Rolandic area.—72. Fisher, *New York Med. Journ.*, April 16, 1898.—73. King, *Chicago Clinical Review*, April, 1897. Glioma.
- Frontal Region.*—74. Schulze, *Deutsche Zeitschrift für Nervenheilkunde*, 1896. Gliosarcoma of right frontal lobe. Operation interrupted owing to hemorrhage. Death next day.—75. Patel, *Lyon Méd.*, xciii, 830. Sarcoma.
- Parietal Region.*—76. Nonne, *Neurolog. Centralblatt*, 1899, p. 229. Subcortical sarcoma, left paracentr. lobe. Death 5 months.
- Cerebellum.*—77. Scheede, *Deutsche med. Wochenschrift*, 1900, No. 30, Cystic glioma in worm. Death.—78. Saenger, *Neurolog. Centralblatt*, 1899, p. 1117. Tumor between pons, medulla and cerebellum, slight improvement.—79. Saenger, *ibid.*, Partial recovery.—80. Joffé, *Deutsche med. Wochenschrift*, 1897, No. 5. Fibrosarcoma of right cerebellum. Operation interrupted on account of collapse. Death in 12 hours.
- Region Indefinite or Not Stated.*—81. Jolly, *Berl. klin. Wochenschrift*, 1899, No. 29, Glioma, too diffuse for removal. Death, 2½ years.—82. Colman and Ballance, *British Med. Journal*, March 11, 1896, Cystic glioma in centrum semiovale. One cyst evacuated. Death in 10 months.—83. Labbè, *Bull. et Mem. de la Soc. Anat.*, 55x, 9, 702, Glioma.

TUMOR REMOVED. RECOVERY.

- Central Region.*—84. Leszynsky, *Med. Record*, Sept. 28, 1901. Endothelioma of right arm and leg center.—85. Hoppe, *Journ. of the Amer. Med. Assoc.*, Feb. 21, 1901. No recurrence after 7 years. No complete cure. Right foot and arm center.—86. Hoppe, *ibid.* Blood cyst from dura over left leg center. Died 18 months in epileptic attack.—87. Mayde, *Wiener klin. Rundschau*. Small cyst, finger center.—88. Heidenhain, *Archiv für klinische Chirurgie*, 64, p. 848. Subcort. cystic sarcoma in right arm center.—89. Treyer, *Rev. méd. de la Suisse romande*, 1900, 5 and 6. Tubercle, right face and arm area. Slight paresis remaining.—90. Treyer, *ibid.* Tubercle, slight paresis remaining.—91. Krauss, *New York Med. Journ.*, July 30, 1898. Sarcoma, left leg center. Death, 2 weeks.—92. Krauss, *ibid.* Infiltrating glioma at junction of leg and arm center, only partly removed. Death, 3 weeks.—93. Oliver and Williamson, *British Med. Journ.*, Nov. 26, 1898. Sarcoma fissure of Rolando. Recurrence.—94. Oliver and Williamson, *ibid.* Subcortical angioma in lower part of left motor region.—95. Stieglitz, Gerster and Lilienthal, *Centralbl. f. Chirurg.*, 1897. Sarcoma. left motor area, recovery.—96. Duncan and Maylord, *Glasgow Med. Journ.*, 1897. Sarcoma of right motor area, improvement.—97. McBurney, *Med. Record*, 1896. Cysts in arm center. Improvement, then death in 6 weeks due to recurrence.—98. Seydel, *Centralbl. f. Chirurg.*, 1896. Fibroma in dura opposite left arm center. Recovery with paresis in leg.—99. Hirsche, *Centralbl. f. Neurol.*, 1896. Diffuse sarcoma of left motor

zone. Partial excision, slight improvement.—100. Devic and Courmont, *Rev. de Méd.*, 1897. Glioma.—101. Sick, *Deutsche med. Wochenschrift*, 1897, No. 2. Tubercle at lower end of gyrus postrolandicus.—102. Cabot, *Bost. Med. and Surg. Journ.*, June, 1897. Cyst punctured twice in 1¼ years.—103. Champonniere, *Wiener klin. Rundschau*, 1897, No. 49. Sarcoma of central convolutions, improved.—104. Krönlein, *Beiträge z. klin. Chir.*, 1896, p. 253. Tubercle in left arm center.—105. Czerny, *Deutsche med. Wochenschrift*, 1896, No. 11. Cystic sarcoma. motor area.—106. Morison, *British Med. Journ.* Subdural cyst, motor area.—107. Voisin, *XII. Internat. Med. Congr.*, Aug. 24, 1897. Tumor of central convolutions.—108. Washburn and Lane, *Clin. Soc. of London*, March 17, 1897. Endothelioma, left motor region.—109. Friedlander and Schlesinger, *K. k. Gesellsch. f. Aerzte in Wien*, Jan. 21, 1898. Gumma, lower end of anterior central lobe.—110. Doyen, *27. Congress der deutsch. Gesell. f. Chir.* Tumor of central convolution.—111. Carriere, *Congrès de Neurolog. Toulouse*, Aug., 1897. Neuroglioma of motor area.—112. Eliot, *Boston Med. and Surg. Journ.* Cyst in lower arm center, evacuated. Death in 3 months.—113. Bonhoeffer, *Monatschr. f. Psych.*, 1898, No. 3. Glioma, upper one-third of right central. Death, 3 months.—114. Dallas and Mongeri, *Gaz. med. d'Orient*, 1897. Sarcoma, lower end of the left postcentral convolution. Death in 4 months of cachexia.—115. Zenner, *Transactions of the American Neurological Association*, May, 1897. Tumor of central convolution. Death 9 months after second operation.—116. Abrahams and Tait, *Occid. Med. Times*, June, 1896. Recovery.—117. Armstrong, *Montreal Med. Journ.*, May, 1896. Cyst, recovered.—118. Braymer, *Charlotte Med. Journ.*, Nov., 1896.—119. Corine and Doyen, *Bull. de la Soc. d. l. Anat. de Paris*, Dec., 1897. Improved.—120. Crowley, *Transact. Medical Soc. of Calif.*, 1898. Sarcoma, improved.—121. Dreyfus, *Lyon Méd.*, 1898. Gumma, improved.—122. Eskridge and Parkhill, *Méd. News*, 1896. Cyst, improved.—123. Eskridge and Parkhill, *ibid.* Angioma, improved.—124. Grisson, *Neurol. Centralblatt*, 1898. Cyst, recovered.—125. Hossler, *Neurol. Centralblatt*, 1897. Improved.—126. Mareau, *Arch. med. d'Angres*, 1897. Improved.—127. Mayer, *Annals of Surgery*, 1898. Cyst, improved.—128. Nedwill, *Lancet*, 1898. Hydatid, improved.—129. Stratton and Crowley, *Pac. Med. Journ.*, 1898. Sarcoma, improved.—130. Tauber, *Arch. de Psych. russ.*, 1896. Improved.—131. Putnam and Richardson, *Boston Med. and Surg. Journ.*, cxi, p. 129. Sarcoma, motor area, not found at first but removed at second operation at same site. Death, 6 months.—132. Gibson, *Trans. of Med.-chirurg. Soc. of Edinburgh*, N. S., xv, p. 27. Glioma, motor area.—133. Sinkler, *Journ. of Nervous and Mental Disease*, xxi, p. 796. Trephined at right facial center, not found, but found and partly removed at second operation. Death, 1 month.—134. Morestin, *Bull. et Mem. de la Soc. Anat.*, 65 i, 249. Sarcoma of cranium, pressing on motor area.

Frontal Region.—135. v. Wagenburg, *Psych. en neurol. Blad.*, 1898. Sarcoma, recovery with slight paralysis.—136. Esteves, *Prog. Med.*, Dec. 23, 1899. Hydatid cyst, recovery.—137. Graff, *Deutsche med. Wochenschrift*, 35, 1899. Cyst, recovery after evacuation.—138. Thomas and Keene, *Amer. Journ. of the Med. Sciences*, Nov., 1896. Gliosarc. recovery with facial paresis and amaurosis.—139. Rossolimo, *Arch. f. Psych.*, xxix, p. 528. Cystic gliosarcoma, death 10 months from recurrence.—140. Naumann, *Hygiea*, 58, p. 155. Sarcoma; periost. cranial, pressing on frontal lobe.—141. Carle, *Rev. de Chirurg.*, Feb., 1899. Sarcoma, recovery.—142. Tubenthal, *Deutsche med. Wochenschrift*, xxv. Cyst, evacuation, recovery.—143a. Fescher, *Deutsche med. Wochenschrift*, xxiv, 52. Gumma, recovery.—143b. v. Bergman, *Berliner klin. Wochenschrift*, 38, p. 219. Tumor, frontal region; recovery.

- Parietal Region.*—144. Clarke and Lansdowne, *British Med. Journ.*, April 19, 1901. Two tumors (encaps. sarcoma) removed within a few weeks.—145. Heidenhain, *Arch. f. klin. Chir.*, vol. lxxiv, p. 848. Tubercle, paracentr. lobe, recovery.—146. Bayerthal, *Münch. med. Wochenschrift*, 1899. Tubercle, paracentral lobe, much improvement.—147. Richardson, *Boston Med. and Surg. Journ.*, 1896. Removed on second trephining.—148. Brissaud, *Bullet. de Chir.*, May, 1896. Tubercle posterior end of the left Sylvian fissure.—149. Starr, *British Med. Journ.*, Oct. 16, 1897. Fibrocyst, inferior parietal lobe. Recurrence in 4 months.—150. Mills and Keene, *Journ. of Nervous and Mental Disease*, 27, p. 244. Endothelioma, superior parietal convolution.—151. Pitt and Lane, *Clin. Soc. Transact.*, 29, p. 164. Sarcoma, recovery.—152. King, *Chic. Clin. Rev.*, April, 1897. Sarcoma, left superior parietal region, recovery.—153. Walton, *Boston Med. and Surg. Journ.*, 144, p. 205. Subcortical parietal cyst, recovery.
- Temporal Region.*—154. Hoppe, *Journ. of Amer. Med. Assoc.*, Feb. 21, 1901. Cyst, first temp.-sphenoid lobe. Recovery with slight aphasia.—155. McCosh, *Med. Record*, 1896. Angioma of pia in left temporal region. Complete recovery.—156. Broca, *Centralbl. f. Chir.*, 1897. Neuroglioma of left temporal lobe. Cure.—157. Hobart, *K. k. Gesell. f. Aerzte in Wien*, June 17, 1898. Gumma, right temporal region.—158. Barr and Nicoll, *British Med. Journ.*, Oct., 1897. Sarcoma in temporal region. Death in 2½ months.—159. Nicoll, *Lancet*, Oct. 29, 1898. Sarcoma originating from middle ear; intracranial portion removed. Death in 2½ months.
- Occipital Lobe.*—160. Hoppe, *Journ. of Amer. Med. Assoc.*, Feb. 2, 1901. Glioma, left occipital lobe. Well after 3 years.—161. Kocher, *Schweiz. Correspbl.*, 27, p. 397. Osteoma growing from occipital bone; recovery.
- Deeper Parts of Brain.*—Heidenhain, *Archiv f. klin. Chir.*, vol. lxxiv, p. 848. Melanosarcoma of tela choroid and ependyma of descending cornua. Entire right temporal lobe extirpated.—163. Bastianelli, *Boll. de la Soc. Lancis*, p. xv, 1. Sarcoma of anterior fossa pressing eyeball forward.
- Cerebellum.*—164. Schede, *Deutsche med. Wochenschrift*, 1900, No. 30. Glioma, recovery.—165. Collins and Brewer, *Med. Record*, May 15, 1897. Subcortical right cerebellar tubercle, partly removed. Death, 2½ months.—166. Guthrie, *Practitioner*, 1898. Cerebellar cyst, improved.—167. Moran and Kerr, *Virg. Med. Semi-monthly*. Cyst, improved.—168. Gibson, *Transac. Med. Chirurg. Soc., Edinburgh*, N. S., xv, 27. Fibrosarcoma cerebellum, removed; recovery.—169. Murri, *Lancet*, 1897. Fibrosarcoma, left cerebellum. Incomplete removal, slight improvement.—170. Parkin, *British Med. Journ.*, 1896, p. 1776. Glioma; recovery.—171. Bevor, Ballance and Lumm, *Brain*, 1897. Fibrosarcoma, anterior part, right cerebellum.—172. v. Bergmann, *Berliner klin. Wochenschrift*, 38, p. 219. Cerebellar cyst; recovery.
- Region Indefinite or Not Stated.*—173. Ziehe and Roth, *Deutsche med. Wochenschrift*, 1897, 19. Perithelioma.—174. Weissgerber, *Münch. med. Wochenschrift*, 1896, No. 16. Cyst in cortex.—175. Kosynski, *Medyzyna*, 1898, No. 1-3, 5.—176. Lundmark, *Ups. Lok. For.*, 1898. Cancer, not improved.—177. O'Hara, *Australas. Med. Gazette*, 1898. Hydatid, recovered.—178. Vance, *Australas. Med. Gazette*, 1898. Hydatid, improved.

VI. TUMOR REMOVED, DEATH.

- Central Region.*—179. Hirschfelder, *Pac. Med. and Surg. Journ.*, April, 1896. Glioma of right post-central gyrus. Death, 7 days.—180. Breganski and Wrzesmowski, *Centralbl. f. Chir.*, 97. Sarcoma of left Rolandic area. Died of meningitis.—181. Schulze, *Deutsche Zeitschrift f. Nervenheilkunde*, 1896. Sarcoma of left anterior central convolution.

Death of hemorrhage.—182. Stieglitz, Gerster and Lilienthal, *Centralbl. f. Chir.*, 1897. Sarcoma in left arm, face and speech center. Death from hemorrhage.—183. Ballance, *Med.-Chir. Transact.*, vol. lxxix. Cystic angiosarcoma, left arm center. Death due to hemorrhage. Had been evacuated four times and removed once before.—184. Dinkler, *Wanderversamml. der Südwest-deutschen Irrenärzte*, May 22, 1899. Fibroma, death in 12 hours.—185. Booth, *Transactions of the American Neurological Association*, May 6, 1897. Tumor, left leg center. Death in 4 hours from hemorrhage.—186. Ziehen, *Zeitschr. f. prakt. Aerzte*, 1898, No. 1. Fibroma, left motor zone. Death, 8 days from meningitis.—187. Hawkes, *Intercolon. Med. Journ.*, Dec., 1897. Hydatid cyst, left fissure of Rolando. Death in 3 days.—188. Schnitzler, *Centralbl. f. d. g. Therap.*, Feb., 1898. Tubercle, right arm center. Death in 26 hours of edema of brain.—189. Taylor and Eliot, *Boston Med. and Surg. Journ.*, 1896. Endothelioma.—190. Starr, *British Med. Journ.*, Oct. 16, 1897. Sarcoma of left upper motor area, death 6 hours, hemorrhage.

Frontal Region.—191. Taylor, *Boston Med. and Surg. Journ.*, Jan., 1896. Endothelioma left frontal region. Death in several hours.—192. Starr, *British Med. Journ.*, Oct. 16, 1897. Cyst, right upper frontal region, removed, recurred; infection with death after second operation.—193. Starr, *British Med. Journ.*, Oct. 16, 1897. Cyst, frontal region, evacuated. Death in 6 days.

Parietal Region.—194. Rychlensky, *Medyzyna*, 1897, 1 and 2. Glioma in paracentral lobe. Death two days after evacuation of cyst.—195. Steffen, *Med. News*, lxxvi, p. 175. Tumor in angular, supramarginal and first temporo-sphenoidal lobe. Death in 2 hours.

Temporal Region.—196. Stewart, *Northwest Lancet*, 1897. Sarcoma.

Deeper Parts of Brain.—197. Dinkler, *Wanderversamml. der Südwest-deutschen Irrenärzte*, May 22, 1899. Tumor in white substance of left cerebrum. Death of shock.—198. Krogus, *Rev. de Chirurg.*, 16, p. 434. Endothelioma of middle fossa; death.

Cerebellum.—199. Pershing, *Med. News*, 1898. Glioma.

Region Indefinite or Not Stated.—200. Hitzig, *Therap. Wochenschrift*. Large endothelioma of dura. Death in collapse.—201 and 202. Lundmark, *Ups. Lok. For.*, 1898. Two cases; died soon after operation.

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A CONTRIBUTION TO THE PATHOLOGY OF PARAMYOCLONUS MULTIPLEX (FRIEDREICH'S TYPE).*

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The peculiar type of muscular contractions described by Friedreich under the name of paramyoclonus multiplex is of very rare occurrence. The uncertain mechanism of its production and the controversy attending its classification and position among the motor neuroses warrant a brief review of the literature as an introduction to the clinical and pathological report which is to follow.

Historical. The history of paramyoclonus multiplex began in the year 1881 with the description of a case by Friedreich,¹ presenting the following peculiarities: The patient was a man with lung tuberculosis, aged fifty, who, following a violent fright, developed quick clonic contractions of the muscles of the proximal segments of the upper and lower extremities. The contractions resembled in character those induced by the electric current, the individual muscles springing forward as though excited by an invisible electrode. It was particularly noted and emphasized that individual muscles, such as the sartorius and supinator longus, would spring forward in independent contractions which singly are incapable of voluntary innervation. Moreover, there was an absence of synergetic muscular action, so that the locomotor effect attending such coördinated muscular contraction was absent. When the contractions were very strong, however, a slight movement resulted. Occasionally, owing to the great rapidity of the contractions, the muscle was thrown into a state of momentary tetanus. There was cessation of the movements during sleep. These muscle contractions were increased by nearly all forms of peripheral and mechanical stimuli. They were diminished or entirely inhibited by voluntary movement, so

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that little inconvenience was caused by performing acts of everyday life, in this respect presenting a marked contrast to the co-ordinated movements of cerebral origin. The knee-jerks were greatly exaggerated. The gross motor power suffered no impairment. The electrical reactions were normal. This was essentially true of the psychical and sensory functions. At times painful flexor spasms of the thigh occurred. There were absolutely no stigmata of hysteria.

The peculiar features of this affection, viz., the spontaneous contraction of individual muscles not singly under the control of the will, and the absence of synergetic muscular action in the affected members, were so at variance with the known characteristics of muscular contractions of cerebral origin as to render some other explanation probable. Friedreich, after a careful consideration of all the possible sources, as the brain, spinal cord, nerves and muscles, proposed the hypothesis which referred the symptoms to an *excessive irritability of the anterior horn cells of the cord*. This theory was in harmony with the special clinical features of the case, i.e., the single muscle contraction, the absence of synergetic action, the great exaggeration of the knee-jerks, and their diminution as the motor symptoms subsided, the great susceptibility of the contractions to peripheral stimuli, and their cessation on voluntary innervation of the part.

In the course of a few years the accuracy of Friedreich's observation was confirmed on many sides by notable observers. Among the earlier recorded cases were those of Löwenfeld,² Remak,³ Seeligmüller,⁴ Francotte,⁵ Homên,⁶ Allen Starr,⁷ Bechterew⁸ and Marie.⁹ It was but natural that Friedreich's description, based on one case, should undergo considerable enlargement and some variation, as the literature was enriched by other observers, and that even new forms and types should be added. Thus, in 1886, Seeligmüller described a congenital case; Unverricht¹⁰ in 1891 added a family form associated with epilepsy, and Gucci¹¹ an hereditary form in 1893. Some of the more important semeiological deviations from the original Friedreich type which have been subsequently described are as follows: That the muscular contractions are *not* limited to the proximal segments of the extremities, but that all the voluntary muscles may be involved with the sole exception of those moving the eyeballs.

Voluntary movement has not in all cases seemed to soothe or interrupt the contractions; indeed, the contrary effect has been noted. In some cases peripheral stimuli have failed to increase the symptoms. The knee-jerks, although usually exaggerated, have been found occasionally of normal intensity or even diminished. In two cases they were absent. The electrical reactions have been essentially normal. In a few cases only was a slight qualitative increase noted. Rarely contractions have persisted during sleep. Pains and paresthesias have been observed accompanying and following the paroxysms. Fright as an etiological factor was by no means constant, and a variety of predisposing and exciting causes are on record.

Classification. But a small proportion of the cases now reposing in the literature under the title paramyoclonus multiplex or its abbreviated form, myoclonus multiplex, are typical examples of this affection. Many cases are hardly recognizable if Friedreich's conception is adhered to, some observations having more in common with convulsive tic, *maladie de tic*, chorea and hysteria. In some observations the peculiar features which led Friedreich to separate this from the other motor neuroses, i.e., the isolated contraction of a muscle not singly under voluntary control and the absence of locomotor effect, have been completely ignored. Cases are recorded as paramyoclonus multiplex in which the affection was characterized by convulsive movements of a paroxysmal character very far indeed from the individual shock-like muscle contraction which usually first becomes apparent when the surface of the body is exposed, and which interferes but little with voluntary movement. This statement will receive more weight when the tabulations of Unverricht are considered, who collected and classified the cases recorded up to the year 1891. Of the forty-eight cases collected, Unverricht accorded to only six the dignity of paramyoclonus multiplex, and to eleven cases a resemblance only to the paramyoclonus; eleven cases he considered uncertain, and the remainder were grouped with hysteria, chorea, or the tic forms.

A survey of the more recent literature shows the same confusion to prevail. Nearly all forms of myoclonia in the broadest sense of the term are included under this head, while the pure cases of paramyoclonus form but a small minority.

It must be observed, however, that muscular contractions identical with or closely resembling those of the essential form of paramyoclonus are found associated with or dependent upon other conditions. Thus, in hysteria and the traumatic neuroses, twitchings resembling paramyoclonus are recorded; and as a sequela of joint affections and then presumably of reflex origin. They are also described in conjunction with epilepsy, the so-called myoclonus epilepsy. Rarely muscular spasms of an entirely different type, i.e., the tic convulsif, *maladie de tic*, are accompanied by movements of a paramyoclonic nature.

In the other forms of myoclonia the Friedreich type may be very closely simulated, but attention to the characteristics just outlined usually renders differentiation possible. Dejerine¹² emphasizes the strong resemblance which some hysterical movements bear to this affection. Indeed, some observers, as Moebius,¹³ regard paramyoclonus as a manifestation of hysteria. Moebius, in support of the hysterical nature of the affection, mentions the difficulty incurred in determining the contraction of an isolated muscle not individually under voluntary control when such contractions are lightning-like. This may be true of the periods of great exacerbation, but would hardly be applicable to the whole course of the disease. Furthermore, it is impossible to reproduce these contractions at will, which, according to our present conceptions, would exclude hysteria. Schultze¹⁴ regards the paramyoclonus multiplex as a generalized form of tic convulsif. Many observers, as Oppenheim,¹⁵ Unverricht, Risien-Russell,¹⁶ Allen Starr and Wollenburg,¹⁷ view this affection as an independent form of motor neurosis. Others, as Raymond,¹⁸ would fuse the various myospasms together under a generic term as myoclonia, considering them as allied in nature and differing only in degree, the degenerative tendency representing the common basis and essential factor in the production of them all. This would include the *maladie de tic*, tic convulsif, electric chorea of Henoch-Bergeron, paramyoclonus multiplex, myokymia, and fibrillary twitchings.

Pathogenesis. Diversity of opinion is not confined to the classification of this affection, but the origin and the mechanism of its production are the subject of controversy. Pathological anatomy has thrown no light upon the subject. The theories which today

receive serious attention are those of the cerebral and the spinal origin. Whether the latter consists of a derangement of the motor portion of the reflex arc (Friedreich) or of the sensory portion, as advocated by Vanlair,¹⁹ is secondary to the question at issue.

According to our present knowledge, the evidence is strongly in favor of a spinal origin of the Friedreich type of paramyoclonus multiplex. The electrical stimulation of the motor cortex with the most delicate electrode fails to produce a contraction of an individual muscle. On the contrary, the result is a movement, a synergetic muscular action. We may assume with a certain degree of probability that movements rather than individual muscles are here represented. The medullary centers, on the other hand, have a more individual relation with the respective muscles under their control, although the exact nature of this relation is still a subject under investigation. It will be remembered in this connection, however, that Sano²⁰ advocates an individual muscle representation in the anterior horn cells, based on experimental studies.

Those rare cases in which paramyoclonic twitchings have occurred in the course of organic disease of the cerebral cortex must be considered in this relation. Examples of such are paresis, chronic meningitis with cortical atrophy (Murri²¹), chronic uremic edema of the cortex (Levi and Follet²²), and myoclonus epilepsy (Unverricht, Clark and Prout²³). These cases are by some observers considered confirmative of a cortical origin. If, however, minute pathological changes of the cerebral cortex were capable of inducing this type of muscular contraction, they should be of more frequent occurrence, considering the vast number of such conditions, constantly under observation. In view of this rarity, a coëxisting alteration of the spinal centers is more probable; and in the family affection, characterized by the association of myoclonus and epilepsy, we are dealing with a degenerative affection of the cerebrospinal axis, the epilepsy referable to the upper, the myoclonus to the lower centers. The few cases of hemimyoclonus (Minkowsky,²⁴ Bernhardt,²⁵ and Seeligmüller²⁶), usually cited as evidence of the cerebral origin, weigh but lightly in the balance.

Very interesting and suggestive from an etiological point of view and corroborative of the spinal theory are those cases of

paramyoclonus multiplex occurring in conjunction with articular affections. Chauffard's²⁷ case of hemiparamyoclonus followed directly on an attempt to break up old joint adhesions in the right knee and hip. Levi and Follet report a case of paramyoclonus multiplex complicating spondylose rhizomelique with the usual joint manifestations. The articular muscular atrophies and the old theory offered in explanation, the perversion of the functions of the anterior horn cells (Paget, Charcot) arise in this connection. Raymond²⁸ demonstrated experimentally that if the posterior roots are cut before the articulation is disturbed, this atrophy will not occur. This is almost indisputable proof of its reflex origin. If pathological reflex stimuli from the articulation can affect the trophic function of the anterior cells, is it not reasonable to infer that the motor function may be perverted in a similar manner with a resulting hyperexcitability? It is a common observation that the tendon jerks are exaggerated in muscles which are the seat of this atrophy, the knee-jerks in affections of the knee joint, the Achilles jerk in ankle joint disease. This exaggeration would hardly be expected in atrophic muscle groups unless accompanied by an unusual irritability of the reflex centers. Gowers²⁹ cites an interesting case in this connection. A young man, following an injury to the left knee, developed an acute atrophy of the quadriceps extensor. When seen two years later the atrophy still persisted, the left knee-jerk was exaggerated, and there was in addition a well-marked ankle clonus upon the left side. This same condition was persistent one and a half years later, and with the associated ankle clonus would seem to prove that the functional and trophic changes in the anterior horn cells in joint affections may not only be of long duration, but may extend to neighboring centers. The muscles from a case of articular atrophy were examined by Darkschewitz³⁰. A majority of the muscle fibers were found to have undergone a diminution in size, averaging about half the normal diameter. These atrophic changes in the muscle fibers, in an affection where presumably the trophic function of the anterior horn cells has been restricted by some inhibitory influence, receive additional significance from the muscle changes found in my case of paramyoclonus multiplex, an affection presumably due to an exaggeration of the function of the anterior horn cells. Here the

diameter of the muscle fibers was two or three times in excess of the normal.

As further evidence in favor of the spinal origin of the peculiar contractions under consideration may be mentioned the relationship existing between paramyoclonus multiplex, myokymia and fibrillary twitchings. Between the fascicular contractions and the muscle waves and the individual muscle contractions of paramyoclonus there are transition forms: a series of gradations, so that one condition merges imperceptibly into the other. The myoclonus fibrillaris multiplex of Kny³¹ represents such a transition form. Thus, in one case the affection may bear the stamp of a myokymia, yet simple fibrillary waves occurring and occasionally the whole muscle undulating; so in paramyoclonus certain undulations may occur. Dana,³² in a recent communication, emphasizes the relationship of myokymia and paramyoclonus multiplex. The dependence of myokymia and fibrillary contractions on disease of the lower nervous mechanism is fairly well established, and has been recently discussed by Walton.³³ The etiology in some of the recorded cases (cited by Walton): lead-intoxication (Huber); following in the wake of old poliomyelitis (Williamson, Walton), low grade neuritis (Biancione), sciatica (Gowers), bear no other interpretation. This receives additional weight from the accompanying pains, paresthesias and electrical disturbances not infrequently observed.

There is still another peculiarity observed in these three forms of muscle contraction, and that is the passive attitude manifested by the patient towards them. The muscle contractions and undulations are felt as such, but otherwise cause little or no disturbance, save occasionally a cramp or a pain. This is largely accounted for by the usually complete preservation of the power of performing voluntary acts and the absence of strong convulsive movements of a coördinated type. It is also possible that their origin in the lower centers, far removed from the psychological sphere, may also play a rôle.

Remarks. I. The term paramyoclonus multiplex, or myoclonus multiplex, should be reserved for that form of myospasm characterized by multiple, spontaneous, isolated contractions of individual muscles.

II. This type is peculiar and distinctive, and receives its most logical explanation in a disturbance of the spinal centers.

III. This type should be carefully separated from the cerebral type of the myospasms which are characterized by movements of a more or less coördinated type, as are observed in the *maladie de tic*, *tic convulsif* and the convulsive tremor of Pritchard and Hammond.

IV. The contractions of paramyoclonus multiplex are closely related to the myokymia and fibrillary contractions.

V. Paramyoclonus multiplex may occur as an idiopathic or a deuteropathic affection, in the latter complicating various organic and functional diseases of cerebral and spinal origin.

CASE REPORT.*

These observations were made in the surgical wards of Bellevue Hospital, service of Dr. B. B. Galludet, through whose courtesy I was permitted to study and publish the case.

History. July, 1901. The patient was a man, aged forty-nine years, Hungarian by birth, tailor by occupation. His admission to the surgical ward was for the treatment of a tuberculous affection of the left ankle joint, which was discharging from several sinuses. An operation had been performed upon the joint at the New York Hospital a few months before. This was his sole complaint on admission, and the surgeons discovered by accident the curious muscular contractions with which he was afflicted and which apparently caused him no concern. It was ascertained that these had made their appearance about six months before without pain or paresthesia, first in the upper, soon after in the lower extremities. There had been no preceding illness except the joint affection before alluded to. He was of moderate habits, denied venereal disease, and no family neurotic taint was discoverable, but owing to his long exile from Hungary his knowledge in this direction was imperfect. No member of his family, to his knowledge, had ever had a convulsion. A few months before the onset of the disease, being poor and in feeble health, he had written to Hungary requesting money from his family with which to return home. This had been promised him, but in due time instead of a money order he received a letter containing a refusal. He despaired of ever being able to return to his native land, became sad and depressed, and a few weeks later the twitchings began. As he related this story his eyes filled with tears, and on subsequent allusion to it he always showed pro-

*Read, with presentation of specimens, before the New York Neurological Society, February, 1903.

found emotion. The twitchings had not interfered with the ordinary acts of life, as dressing, walking or holding a newspaper, and had caused him but little annoyance; toward the end of the day he would feel rather fatigued.

Physical Examination. The man is of medium height, well made, body weight well preserved. Facial expression is quiet and melancholy. Skin pallid, mucous membrane a fair color. The face free from tremor or movement of any kind. Dressed and sitting in a chair, as when I first saw him, no abnormal movements of any kind were visible, except an occasional playing of the fingers. Locomotion and station were naturally somewhat interfered with by virtue of the joint affection, but were entirely unaffected by the muscular contractions. The pupils and pupillary reactions were normal, as were all the cranial nerves. No nystagmus. After removal of the clothing, the picture presented by the patient was a strange and unusual one. The muscles of the arms and shoulder girdles, thighs and buttocks displayed the greatest activity, individual muscles springing forward in contraction with the greatest rapidity and variation; sharp, shock-like contractions following one another with regularity and rhythm. The whole body of a muscle contracted as if stimulated by an invisible electrode. These contractions were unaccompanied by any locomotor effect. Occasionally, a barely perceptible flexion, extension or supination resulted from a particularly violent or prolonged contraction. It was further noted that while symmetrical muscles were involved, the contractions were by no means synchronous or isochronous. The movements were all clonic, no tonic contractions being noted. Contractions were noted in the following muscles: Deltoids, pectorals, scapular group, biceps, triceps, supinators, gluteal groups, extensors and flexors of the knee, and the sartorius; an occasional contraction was noted in the forearms and in the calves of the legs. The greatest play of movement was in the proximal segment of the extremities. The abdominal muscles and the diaphragm were quite free. The intensity and rapidity of these contractions could be modified in various ways. Mechanical stimulation of the skin and muscle increased them, while a coördinated act, such as grasping or picking up an object, caused their cessation or a very marked diminution. In walking and standing this diminution in the lower extremities was very noticeable. The contractions were most severe in the recumbent posture, less while standing, and almost disappearing while walking to and fro. An unusual and important peculiarity of these spasms was the contraction of individual muscles, as the sartorius and supinator longus, the isolated contraction of which cannot be produced at will. There were apparently isolated contractions of the brachialis anticus and coraco-brachialis, but this could not be determined with cer-

tainty. The muscular system was quite well developed and showed no evidence of pathological hypertrophy or atrophy. The gross motor function was undisturbed. The myotatic irritability was increased. No electrical examination was made. Sensation was undisturbed. The tendon and skin reflexes were all present and very active; the knee-jerks especially were much exaggerated. Flexor response to plantar stimulation. No stigmata of hysteria.

The man was quiet and uncomplaining and shunned the society of his fellow-patients. He presented no evidence of mental enfeeblement. A few weeks later acute symptoms developed, death ensuing apparently from a generalization of the tuberculous process; as the thoracic and abdominal cavities were not opened, the immediate cause of death must remain unknown. It is interesting to note that the muscular contractions persisted the whole time during which he was under observation, varying, however, in intensity, and, according to the statement of the house-surgeon, were observed for twenty minutes after the respiratory and cardiac functions had ceased. According to the nurse's statement, the contractions ceased during sleep.

Histological Examination. The autopsy was performed forty-eight hours after death. The brain, spinal cord, and the right radial and anterior crural nerves were removed and placed in 10 per cent formalin solution. Portions of the right supinator longus, right sartorius, and left trapezius muscles were also removed.

Brain. Sections were prepared from the Rolandic area on both sides and from the frontal, temporal and occipital lobes, according to the Nissl, Weigert-Pal, Marchi and Van Gieson methods. No evidences of pathological changes were discoverable in nerve structures, meninges, vessels or glia. The outlines of the cortical cells and their processes are well preserved. A few cells are moderately pigmented. The nuclei, nucleoli and their refractive bodies are quite distinct. The nuclear network, however, is broken up and appears granular and homogeneous, remaining unstained or receiving a faint bluish tint. The Nissl granulations are coalescent and rather clumpy, staining intensely blue. The slight alterations in the nuclei and Nissl bodies are dependent on cadaveric changes.

Spinal Cord. Sections were prepared from various levels of the cord by the Nissl, Weigert-Pal, Marchi and Van Gieson methods. They were essentially normal. The spinal stichochromes of the anterior horns were absolutely normal. Cell bodies, processes, nuclei and nucleoli, no excess of pigmentation. The Nissl granulations, while somewhat coarse and rounded, are normal in distribution and arrangement. The vessels of the gray matter of the cord, especially in the cervical and upper dorsal regions, are distinctly thickened. The vessels in the lateral

columns are also slightly sclerosed. This thickening involves chiefly the adventitia; nowhere was obliteration of the lumen observed.

The capillaries of the brain and cord are distended with blood.

Peripheral Nerves. The peripheral nerves, the inter-muscular nerve fibers and the muscle spindles were normal.

Muscles. The muscles were fixed and hardened in formalin, alcohol and Müller's fluid, and stained by the carmine, hematoxylin, Van Gieson and the Marchi methods. The supporting structures (endomysium and perimysium) were normal, as were the blood vessels.

The picture presented by the muscles on transverse section is striking, the diameter of the muscle fibers reaching an unusual size (see Figs. 1 and 2). Of the larger fibers which form the

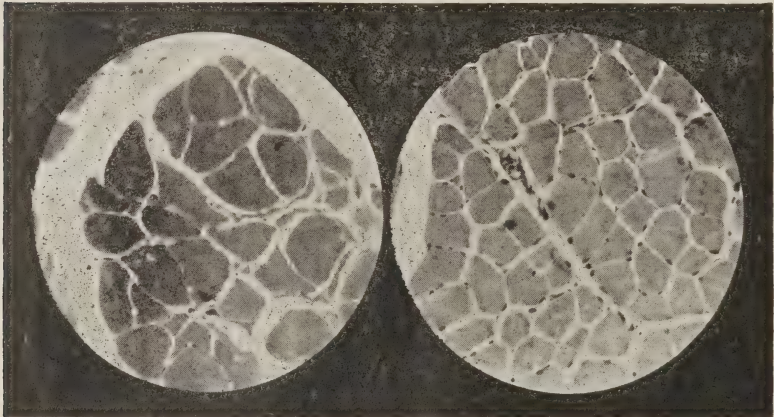


Fig. 1.

Fig. 2.

Fig. 1—Right supinator longus muscle from case of paramyoclonus multiplex.

Fig. 2—Normal muscle. Same enlargement as Fig. 1.

majority in any given field, the diameter averages 150 micromillimeters and, in many, 175 micromillimeters. In contrast to this are very small fibers having crescentic forms or flattened out on the periphery of the large fibers. Of these smaller fibers some are below the normal standard. The fields of Cohnheim are quite distinct. Instead of finding the nuclei of the sarcolemma sheath confined to the periphery of the muscle fiber and just beneath this membrane, as is customary in man, they are found scattered as well between the sarcous elements (Fig. 3). This is not true of all the fibers, but is observed in a large number and in all sections studied. Sometimes as many as three or four nuclei occupied a

central position in a single fiber, usually only one or two. They are identical in staining properties with those of the sarcolemma sheath. On longitudinal section the transverse striation is distinct and well preserved (Fig. 4). In the smaller fibers only is it at times indistinct or absent, these showing in addition a tendency to longitudinal cleaving. The nuclei of the sarcolemma sheath



Fig. 3—Sartorius muscle. Paramyoclonus multiplex. Fibers hypertrophied with centrally situated nuclei.

are increased in number; their structure and size are normal. Small clumps of a dark brown or yellow pigment are observed scattered over both the transverse and longitudinal sections, in the latter especially in relation with the sarcolemma nuclei. No degeneration and no vacuolization of the sarcous substance is present. No degenerations were demonstrable by the Marchi method.

Remarks. The peculiar character of the muscular contractions in the case just described entitles it to a place among those rare cases of essential paramyoclonus multiplex. The histological examination of the spinal cord and biceps muscle from the original Friedreich case was carried out by Schultze.³⁴ The result was an entirely negative one. This and my own case constitute, I believe, the only examples on record of this type of

paramyoclonus multiplex with systematic histological examination. The examination of the nervous system in my case was entirely normal. The muscle fibers, on the other hand, while retaining their normal structure, were considerably hypertrophied.

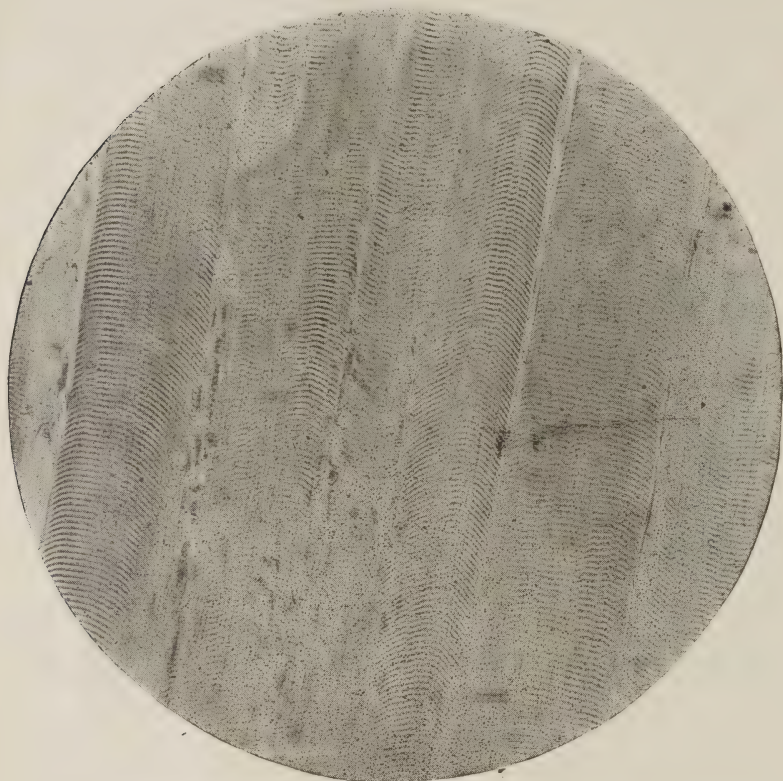


Fig. 4—Muscle showing transverse striations. Paramyoclonus multiplex.

They were twice or three times the size of normal fibers from corresponding muscle utilized as control preparations. Muscle fibers vary considerably in size even under normal conditions. In general, the larger the muscle the larger is the muscle fiber. In man the minimum diameter is given as 10 micromillimeters; the maximum as 100 micromillimeters. In the case under consideration, the larger proportion of the muscle fibers on transverse section averaged a diameter of 150 micromillimeters, many measur-

ing 175 micromillimeters. This increase in size of the fibers was not universally distributed over the microscopic field. In round numbers, two thirds may be said to have presented this change. Many of those remaining were below the normal standard, so that the contrast was very evident.

Another peculiarity was the presence of sarcolemma nuclei between the sarcous elements. This is a peculiarity of the so-called red fibers of the lower vertebrates, a form of muscle which is more active, and capable of more prolonged contraction, than the white fibers which constitute human muscle, although such red fibers are scattered in very small number throughout the muscular system in man.



Fig. 5—Sartorius muscle from a case of chronic progressive chorea [control preparation]. Same enlargement as Fig. 3.

It is interesting and significant to observe that changes similar to those just mentioned have been described in cases of myotonia congenita (Erb,³⁵ Dejerine and Sottas³⁶), the hypertrophic stage of the muscular dystrophies, and in those rare cases of true muscular hypertrophy, of which examples have been described by Friedreich,³⁷ Auerbach,³⁸ and Berger³⁹. If such alterations in the size and nuclear constituents of the muscle fiber were simply the result of overaction, and secondary only to violent and prolonged

muscular action, they should occur in long-standing spasmodic affections of cerebral origin. In one of the cases used as a control preparation in the present study, a chronic progressive chorea of fifteen years' duration, the muscles were found absolutely normal (Fig. 5). It is suggestive that in the muscular dystrophies and hypertrophies and in myotonia and in paramyoclonus multiplex, which represent obscure affections of the muscular and neuromuscular systems respectively, muscle changes essentially similar should be encountered. Hajos' ⁴⁰ case of paramyoclonus multiplex with the electrical reactions of Thomsen's disease is of interest as furnishing clinical evidence of this relationship. We are far from understanding the true significance of the relations existing between the peripheral motor neurone and the muscle fiber, tissues having a different structure and derivation and yet intimately related; the fate of the one resting entirely upon the integrity of the other—the so-called *trophic function or influence of the anterior horn cells*.

In articular muscle atrophies, this trophic influence in some obscure way appears to undergo a reflex inhibition. In the Darkschewitz case the muscle fibers had suffered a reduction of one half their natural size.

As this trophic influence may under certain conditions be so perverted as to cause disintegration or atrophy of the muscle fibers, it is reasonable to assume the possibility of an increased trophic function with a resulting hypertrophy. If the Friedreich theory of the irritability of the anterior horn cells is accepted in explanation of the motor phenomena of paramyoclonus, these muscle changes may well represent a concomitant increase of the trophic function of these same cells, and this would constitute important objective evidence of the spinal origin of paramyoclonus multiplex.

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Society Proceedings.

NEW YORK NEUROLOGICAL SOCIETY.

March 3, 1903.

The President, Dr. Pearce Bailey, in the chair.

Tabes Associated with Hemiplegia.—Dr. Joseph Collins presented a man, forty-two years of age, a bar tender by occupation. When twenty-four years old he had had a chancre, for which treatment was given. A year later an iritis developed, and disappeared after two months' treatment. There was nothing further until eight years ago, when he was seized with an attack of vertigo, which was associated with an aphasia lasting several days. There was no paralysis. Four years later there was a similar attack associated with vomiting. Last August the man fell in an attack in which he was only partially unconscious. Examination showed a right-sided hemiplegia, and this had continued ever since. On coming to the hospital, there was found in addition a very marked atrophy of the right shoulder and an absence of both knee-jerks. The pupils were small and regular, and reacted to accommodation but not to light. There were incontinence of urine and manifest ataxia. The case was presented as one of tabes, the latter probably antedating the attack of thrombosis from which the hemiplegia originated. Coincident with the attack of cerebral thrombosis there must have been an obliteration of one of the cornual branches of the anterior spinal artery. In reality, this case presented three conditions: tabes, cerebral thrombosis and a destructive poliomyelitis of very limited extent. Tabes and hemiplegia were rarely associated.

Dr. J. Ramsey Hunt said that he had examined this case in the City Hospital. He had been doubtful about its being an anterior horn disease, and inclined to the opinion that there was a plexus lesion.

Dr. M. G. Schlapp said that in some of these cases of hemiplegia certain groups of muscles were markedly atrophied. The electrical examination should decide whether the case was of central or peripheral origin.

Dr. J. Fraenkel said that in only a few of the cases of tabes with hemiplegia had the knee-jerk returned, and this had only been temporary.

Dr. C. L. Dana said that he had had a short time ago under observation a man with tabes whom he had seen the very day of the occurrence of the stroke. There had not been any return of the knee-jerk, and he thought this could only occur in the early stage.

Dr. Collins said that there had been no suspicion of the existence of tabes before he had examined this man, and he had been deeply interested in the presence of the marked muscular atrophy.

A Case of Major Hysteria (?).—Dr. Collins then presented a woman, forty-four years of age, a cook by occupation. There was no history of alcoholism or of venereal disease. The present trouble began four years ago with an involuntary twitching of the hands and a loss of sensation in the hands and arms. This loss of sensation had gradually spread over the entire body. She had no pain, but felt tired and walked with difficulty. The gait was not strictly ataxic, but was rather shuffling and choreic. There was a peculiar condition of the left lower extremity, the lower third being shrunken without any hardening or thickening of the skin. Examination showed universal analgesia and more or less thermal anesthesia

and analgesia. The knee-jerks and ankle jerks were exaggerated. There was slight disturbance of speech but not of intellection. She presented none of the ordinary stigmata of hysteria, and the color fields were of normal size. The woman had not improved under hospital treatment. According to the history, her father and sister were similarly affected, but the former lived to be over seventy years old. The case was presented without a positive diagnosis, because it did not seem to Dr. Collins to correspond to hysteria, syringomyelia or Huntington's chorea.

Dr. Schlapp was of the opinion that the case was one of hysteria, because the condition did not correspond to any known pathological lesion. The condition of the leg might have been brought about by a self-inflicted trauma. Two years ago he had presented to the Society a case of supposed atypical zoster, but subsequent investigation showed that the patient had produced the condition herself by means of carbolic acid.

Dr. R. H. Cunningham said he was reminded of a number of cases that he had had in a family in Richmond, Va., in 1893. Choreic symptoms developed between the ages of fifteen and thirty in the father, two sons and two daughters. One of the daughters had hemianesthesia. He would look upon the case just presented as one of adult chorea.

Dr. Collins said that he had been inclined to look upon the case as one of hysteria, yet this diagnosis presented many difficulties. After having observed the patient carefully for several months he was still more inclined to the diagnosis of major hysteria.

Dr. Pearce Bailey said that he thought the ataxic gait was due to the anesthesia, and that the case was one of major hysteria. With regard to the other case, there seemed to be no doubt that the atrophy of the shoulder was distinct from the tabes. A fall on the point of the shoulder was quite frequently the cause of a paralysis with just such an atrophy.

A Case of Spinal Tumor.—Dr. M. G. Schlapp presented a man, forty-four years of age, with a good previous history. About two years ago a peculiar twitching had been noticed in the muscles over the right shoulder, and about two months later he had begun to suffer pain at the root of the neck and extending into the trapezius muscle. After a time the right arm became weak, and subsequently the left side. This weakness was found to correspond to the muscles supplied by the anterior and posterior thoracic nerves on both sides. There was the Babinski symptom on each side. There were analgesia and diminished temperature sense on the left side. The diagnosis made was an extramedullary tumor involving the fifth and sixth cervical roots of the spinal cord, pressing upon the pyramidal tracts, and on Gowers' tracts, diminishing the pain sense and temperature sense. The tumor lay on the right side, and probably involved the motor roots on the other side. He thought an intramedullary tumor could be excluded. The man was to be operated on.

Dr. Pierce Bailey said he had seen this man last Fall, and at that time there was no sensory disturbance, and the pain dated back two years, but with intermissions. He had formed the opinion then that there was a degenerative lesion rather than a tumor.

A Case of Cerebellar Tumor.—Dr. J. Ramsey Hunt reported a case of cerebellar tumor with degenerations of the posterior columns. The subject of this report was a man of forty-six who had been admitted to Bellevue Hospital in July, 1902, with typical symptoms of cerebellar tumor, although the tumor could not be localized. The cerebellar symptoms had begun three months before, and the patient was under observation for three months. During these six months there was a progressive diminution of the knee-jerks and Achilles jerks on both sides. It was thought to be due to degenerations in the posterior columns of the cord, as had been described in connection with tumors in the posterior fossa of the skull. There was very marked choked disk, and the man's mental condition became one of apathy and somnolence. At the autopsy two small tumors were

found, one springing from the dura mater in the median line, and encroaching somewhat upon the right frontal lobe, and the other tumor was in the left cerebellar hemisphere. Both tumors were medium-sized round-cell sarcomata. The spinal cord presented evidence of greatly increased intracranial pressure. Specimens of the spinal cord, both transverse and longitudinal, were prepared, and they apparently showed degenerations of the posterior columns, such as had been described in connection with tumor. The higher levels of the cord were most affected. Lissauer's column was spared, as was the rule. The fibers in Clarke's columns were also usually spared. These degenerations arose from the entrance of the posterior roots into the spinal cord. It was an anatomical fact, that where the posterior roots perforated the dura mater obliquely there was a constriction, and that here the sheath of Schwann was lost. At the same time there was a constriction of the myelin substance, and some of the fibers were deprived entirely of this substance. The speaker then briefly discussed the mechanical and toxic theories, and said that he presented the case as one favoring the mechanical theory.

A Case of Cerebellar Tumor.—Dr. L. Pierce Clark reported the following case: The subject was a man of thirty, who had had both gonorrhea and syphilis, and who used alcohol and tobacco to excess. The present illness began in October, 1901, with headache in the right occipital region. After a time he began to vomit in the mornings, and the right side became weak. Iodide of mercury had been given without benefit. On examination, there was choked disk in the right eye; the sixth nerve was bilaterally weak; the muscles of the right leg were stiff and painful; the head was held stiffly and bent slightly to the right side; all the deep reflexes were exaggerated, particularly on the right side; there was double Babinski; the gait was characteristically cerebellar. A diagnosis was made of a syphilitic tumor in the middle lobe of the cerebellum involving the peduncle. On admission to the Presbyterian Hospital in January, 1903, for operation, an X-ray examination showed the tumor. The man died rather suddenly, and the autopsy disclosed a tumor of the cerebellum. Between it and the fourth ventricle was a large cyst filled with slightly turbid serum. Apparently the growth was a syphilitic gumma that had undergone syphilitic and calcareous degeneration.

A Case of Spindle-Cell Sarcoma of the Cerebral Dura.—Dr. B. Onuf reported this case and presented a specimen. The subject of the report had been first seen on May 2, 1902, and at that time complained of a shooting pain in the left ear and in the teeth, and of attacks of dizziness. There was only slight perception of light; the gait was staggering, there were no marked changes in sensation or in the reflexes; there was typical choked disk. A diagnosis of cerebellar tumor was made. Subsequently the man developed slight paresis of the left facial nerve with fibrillary twitchings of the muscles in this region, and the hearing on the left side became greatly impaired. In walking, the man deviated to the right. He died in December, and at the autopsy a tumor was found at the base of the left cerebellar hemisphere. It was a spindle-cell sarcoma. This could have been enucleated had it been in a more accessible situation.

Dr. Schlapp said that about a year ago he had presented to the New York Pathological Society a case similar to the one presented this evening by Dr. Hunt showing degeneration of the spinal cord. The specimens from his case had been counterstained with acid rubin and the sheath of Schwann was stained. In all of the cases the degeneration extended to this sheath, especially in the cranial nerves, the trigeminus, the glossopharyngeus and the vagus nerves. He had expressed the opinion at that time that the sheath of Schwann might have some protective influence. He did not think the case tended to disprove the toxic theory. There were no sensory symptoms in the beginning, and probably the degeneration was at first confined to the fibers passing from the posterior columns into the

gray matter. The poison appeared to be selective in its action, involving only the reflex fibers. The degeneration was particularly marked in the sensory nerves.

Dr. W. M. Leszynsky said that he had been interested in the fact that respiratory failure had preceded cardiac failure in Dr. Clark's case. One of his own patients had died upon the operating table just as the cerebellar abscess was reached. The heart continued to beat for eight or ten minutes after the failure of respiration. If degeneration existed in the posterior columns it must act chiefly on the third or fourth lumbar segment in order to produce loss of knee-jerks; how, then, would one explain those cases in which the knee-jerk was absent for a time, then became well marked, and was alternately present and absent?

Muscle Tonus and Tendon Phenomena; Their Relationship and Interpretation.—Drs. J. Fraenkel and Joseph Collins presented this paper, which was read by Dr. Fraenkel. It was based upon tonometric examinations of 230 patients. The authors stated that at the present day the view most generally held was that the tendon phenomena were expressions of muscle tonus. General clinical experience showed a definite relationship between tendon phenomena and the tonicity of muscles. Hypertonia, next to exaggeration of the tendon jerks, was a symptom of disease of the pyramidal tracts. The tonometer used in this investigation was the invention of the house physician of the Montefiore Hospital. A total of 554 registrations had been made. The 230 cases were distributed as follows: Apparently healthy, 32; cases of tabes, 25; of organic disease of the nervous system, 33; functional disease of the nervous system, 40; pulmonary disease, 70; heart disease, 12, and various other chronic affections, 18 cases. There were 71 registrations classed as hypertonia. It was found to be frequently, although not always, associated with exaggerated Achilles reflexes. Hypotonia was found to be frequently associated with loss or diminution of the tendon phenomena. There was a comparatively small number of neurogene hypotonias in proportion to the cases of general hypotonia. Neurogene hypotonia and absence of diminution of the tendon reflex were found frequently associated. Neurogene hypotonia with exaggeration of the tendon reflexes was present in nine per cent. It was concluded that hypotonia with exaggeration of the tendon reflexes was present in nine per cent. It was concluded that hypotonia produced by damage of the ascending tracts of the spinal cord was accompanied by loss or absence of the tendon phenomena in every instance. In the cases of hypertonia absent or diminished reflexes were present in six per cent of the registrations. In the cerebral group there was found a larger percentage of hypotonias than of hypertonias. In the spinal group 100 per cent gave exaggerated tendon reflex and 95 per cent showed hypertonia. It had been learned that whenever the neurogene tone was markedly increased or decreased the tendon jerks were increased or decreased correspondingly. The authors concluded that disease of the posterior tracts caused hypotonia, and disease of the pyramidal tracts caused hypertonia. There was a large group of cases giving tonicity and normal tendon phenomena.

Dr. G. L. Walton, of Boston, said that in the exhaustive discussion on reflexes and tonus opened by Crocq in 1901, the reader had reviewed the clinical and experimental evidence which tended to show that the reflexes generally corresponded to the tonicity. The exceptions to this rule he explained as illustrations of the fact that the centers for tonicity (at the cortex) and those for the deep reflexes (in the basilar region) were separate. This explanation was not directly discussed, though the limitation of these functions to the regions indicated was seriously questioned. Drs. Fraenkel and Collins had reinforced in a most convincing way the results of Crocq, though their explanation of the exceptional cases differed from his materially. The question was too complicated to be discussed in its en-

tirety, especially in the absence of exact knowledge as to the seat of either the tonus or the reflexes, but such work as had been reported by Drs. Fraenkel and Collins furnished one of the definite steps by which one might gradually mount to a knowledge of this complex subject. We had been so long imbued with the notion that in man, as in the frog, the spinal cord was the center for reflexes and tonus that it was difficult to transfer our study of these functions to the brain. Even in case of disturbed reflex in cerebral disease we were still apt to think of the function of the brain as limited to the withdrawal, or the increase of influence upon the cord, through the pyramidal tract. For a long time the hypotonicity and loss of reflex sometimes found at the onset of cerebral hemorrhage was attributed to the shock conveyed to the cord. At the discussion already alluded to the disputants, while recognizing the importance of the cerebral influence, were by no means in accord with Crocq in limiting the reflex centers and the centers of tonicity to the brain. Dr. Walton said he had several times taken occasion to suggest that, instead of speaking of lower centers controlled, or inhibited, by higher centers, inhibited perhaps in their turn by still higher centers, we should recognize the combined action of all centers, cortical, basal and spinal, allotting to certain regions a predominating reflex function, liable to transference to a lower level on gradual withdrawal of upper level influence. According to this view each reflex movement might be regarded as a resultant of the activity of various loops of different lengths, connected with each other both laterally and vertically. The more he had observed the reflexes in cerebral disease the more he was inclined to accord to the cortex the chief rôle in the deep, as well as the superficial reflexes, and in the tonus. It was difficult, for example, to explain on any other basis the absence of deep and superficial reflexes in the following case: The patient was unconscious and hemiplegic, with a temperature of 105° F., and all reflexes absent up to the time of his death. The Kernig symptom was present, showing, probably, comparative hypertonicity of the hamstrings, but the extremities were otherwise obviously hypotonic. Autopsy showed pneumococcus meningitis of the convexity with encephalitic invasion of the cortex. In other cases of meningitis the reflexes were frequently preserved, but in one case of meningitis coming under his observation, in which both brain and cord were affected, during the stage of rigidity and obvious hypertonicity the reflexes were absent, but reappeared as relaxation set in, first on the side first relaxed. One must evidently look farther than to separation of the centers to explain such conflicting phenomena; in fact, different cases might require different explanations. Possibly it was partly a question of comparative tonicity. In the last case, for example, the hamstrings might have possessed hypertonicity so far in excess of the hypertonicity of the quadriceps femoris as to "snub" the knee-jerk. In long standing infantile cerebral hemiplegia with contracture the deep reflexes were often absent, though hypertonicity was apparent. Doubtless in some of these cases the contracture was in such position as to put the tendons on too great or too little stretch, thus mechanically preventing the reflex. The combination of exaggerated reflexes with hypotonicity was sometimes seen in Erb's syphilis of the cord, especially in the early stages. In a recent conversation with Dr. Courtney he had suggested that the affection about the fibers of the pyramidal tract was sufficient to impede the transmission of voluntary impulse and of tonicity from the cortex, but the reflex stimulus passed the more rapidly, as in neurasthenia, uninhibited by the volitional mechanism. Though not sure that he had thoroughly grasped the explanation of the readers on this point, Dr. Walton felt that they had proceeded along logical lines.

Dr. C. L. Dana thought the authors had established quite clearly the relations of hypertonia to the reflexes, and had given to the neurological world tangible working data. He had not fully understood the explanation of the effect of the brain on hypotonus and hypertonus. Personally, he

made use of a working hypothesis which fitted in very well with the explanation given in this paper. In all the sudden insults to the brain occasioned by hemorrhages producing profound hemiplegia there was always absence of the deep reflexes on the paralyzed side, and with it no doubt hypotonia; whereas there was not this loss of reflexes on the non-paralyzed side. After a time this absence gave place to an increase of reflex. If, however, the hemorrhages were more posterior and involved the sensory sphere, there was more definite and more prolonged absence of reflexes.

Dr. B. Onuf referred to a case in which a tumor of the hip and shoulder centers was diagnosed, and the tumor removed, with the result that a very marked hypotonia developed in the paralyzed extremity after the operation, and was present even two months afterward.

Dr. Collins said that Dr. Fraenkel was satisfied with having established the relationship between tonus and reflexes, and they were both particularly gratified with Dr. Dana's statement that in his cases of cerebral hemiplegia he had observed that the farther posteriorly the lesion extended, either into the sensory cortex or the sensory representation of the capsule, so in proportion was the existence of hypertonia or the lateness with which it appeared. This clinical experience fortified very greatly the position taken in the paper.

Dr. Fraenkel said that he too had been delighted at the confirmation of their position by the very large clinical experience of Dr. Dana. If their observations were correct they should be of value in connection with diagnosis of pure lesions of the pyramidal tract associated with hypertonia and of the posterior tract with hypotonia.

PHILADELPHIA NEUROLOGICAL SOCIETY.

March 24, 1903.

The President, Dr. H. A. Hare, in the chair.

A Case of Progressive Bulbar Palsy.—This case was exhibited by Dr. T. H. Weisenberg. The patient had presented herself at the nervous dispensary of the Polyclinic Hospital three weeks previously with the statement that four weeks before coming to the clinic she had suddenly lost the power of speech. This she attributed to the wearing of false teeth. She protrudes the tongue in a straight line and moves it from side to side. There is a fine fibrillar tremor in the tongue. She can not pucker up the lips. There seems to be no involvement of the upper distribution of the seventh nerve. There is no apparent atrophy of the muscles of the tongue or mouth. She has typical bulbar speech. She swallows with difficulty. There is preservation of the pharyngeal reflex. The jaw-jerk is present. The knee-jerk is normal. The gait and station are normal. On laryngoscopic examination, no gross change is observed.

Dr. Charles W. Burr called attention to the fact that it was rare in a case of this kind to find such great involvement of the speech with such slight palsy of the lips as was present in this woman. As a rule in disease of the bulb when the speech becomes so bad as not to be understood there is marked palsy of the muscles of the tongue and lips.

Dr. William G. Spiller said that a curious feature about this case was that the loss of speech came on suddenly. This he thought was an error of observation on the part of the patient, although such a loss may occur suddenly, probably from hemorrhage. This he considered a case of true bulbar paralysis, not a case of pseudo-bulbar paralysis.

A Case of Tuberculous Meningitis with Secondary Infection.—This was reported by Drs. S. S. Kneass, W. F. Hendrickson and Joseph Sailer. The patient, a man aged twenty-nine years, was admitted to the Hospital of the University of Pennsylvania, March 15, 1902. His illness began with headache, nausea, vomiting, diarrhea and soreness and stiffness in the back. He had been treated for typhoid fever until March 8. He had gradually improved with the exception of the pain in the head. On March 12, in the third week of the disease, he became delirious.

Examination on admission showed moderate leucocytosis, diazo reaction negative, Widal reaction negative. On account of the cerebral symptoms, lumbar puncture was performed, and examination of the fluid showed the presence of a diplococcus. The patient was apathetic, but intelligent, answering properly, and did not complain of pain. Both pupils reacted normally. There was general hyperesthesia to all forms of sensation. Respiration was abdominal and the abdomen was retracted. There was a loud, short systolic murmur. Examination of the lungs was negative. The knee-jerks were absent, but there was no spasticity. The patient developed edema of the lungs, and on the second day after admission became comatose. Under the use of digitalis the edema of the lungs cleared up, but he died on the 19th.

Autopsy.—The heart was not enlarged,—no valvular lesion. The lungs were congested and edematous. The kidneys normal. The brain showed marked congestion, and at the base a puriform exudate into the meninges. Numerous tubercles were found on the meninges, and smears showed a large number of tubercle bacilli. The lungs presented a number of microscopic miliary tubercles. The meninges also showed marked infiltration of the lymphoid cells. The interesting feature of the case was the mistake in diagnosis based upon the presence of the *Micrococcus tetragenus* found in the spinal fluid. As far as the authors were aware this was the only case in which this had been found. The primary focus of the miliary tubercles was not found.

Dr. Alfred Gordon said that the most recent cytological studies of the cerebrospinal fluid threw considerable light upon the question of meningitis. These tend to show that lymphocytosis of the cerebrospinal fluid is a characteristic feature of an inflammatory condition of the meninges. It would be, therefore, interesting to know the result of the cytological examination in Dr. Sailer's case.

Hemiplegia without Gross Lesions in a Case of Plumbism and Nephritis.—This paper was read by Dr. Charles W. Burr. The patient was a man aged fifty-five years, admitted to the hospital February 3, 1903, in an unconscious condition. He had worked in the salts of lead. There was palsy of the right arm and leg and of the right side of the face. Both knee-jerks were fairly active. There was some extension of the great toe on stroking the sole of the foot on the paralyzed side. The man was stupid and delirious but not absolutely unconscious. On the second day, he became completely unconscious, and seventy-two hours after the onset of the paralysis, which had been sudden, he died. There was a distinct lead line on the gums, hyaline casts and a line of albumin were found in the urine. No gross lesion was found in the brain, and the only lesion discovered was chronic inflammation of the kidney. There was no arterial sclerosis in the brain or other part of the body.

A Medullary Nucleus not as yet Described.—A brief report of this was made by Dr. E. L. Mellus, of Baltimore.

Dr. Joseph Sailer thought that Dr. Mellus had proved the point for which he contended. What he had shown was certainly a nucleus. He congratulated the Society on the honor done it by Dr. Mellus in making the first report of the discovery to it.

Dr. William G. Spiller said that there was a distinct group of cells in the area referred to. There is also a similar group of cells in the human

brain, although in the dog the cells are much larger. He raised the question whether this might not be a part of Dieters' nucleus. Whether or not it is to be considered a part of Dieters' nucleus, it is a distinct group of cells and has as much right to be considered a special nucleus as von Bechterew's nucleus which also may be regarded as a part of Dieters' nucleus. The determination of this question will rest with the discovery of the function of these cells.

A Case of Complete Brachial Monoplegia.—This case was presented by Dr. G. M. Dorrance. The patient was a man aged twenty-four years, who had come to the nervous clinic of the Polyclinic Hospital. In July, 1902, while working in a shaft an iron bucket fell, striking him on the left shoulder. He was rendered unconscious and remained in that condition for seven days. Since the accident he has not been able to use the left arm. The muscles have gradually wasted and the reactions of degeneration are present. The case was presented especially with reference to the propriety of operative procedure.

Dr. F. Savary Pearce said that he had presented a somewhat similar case about a year ago. There was degeneration-reaction throughout the brachial plexus distribution, and there seemed to be no help for the man, and amputation was finally done. Microscopic examination of the nerves of the brachial plexus showed widespread degeneration. It was Dr. Pearce's opinion that in a case of this kind, where there was so much degeneration and the member was useless, similar operations should be done. There was no delay in healing of the stump in the case Dr. Pearce reported, and the man was relieved of an incumbrance, much to his delight.

Dr. William G. Spiller referred to a case which he had seen in which Dr. Keen had exposed and resected the musculospinal nerve. A year and a half later there was almost complete return of power. In some cases there has been tearing and thickening of the nerves, and resection of the thickened portions and bringing the ends together has been followed by good results.

Periscope.

ARCHIVES DE NEUROLOGIE

(No. 86, 1903, February.)

1. Contribution to the Study of "Folies" by Contagion. G. CARRIER.
2. On Obsessions and Impulsions (Episodic Syndromes in Degenerates).
DR. SOUTZO, JR.

1. *Contribution to the Study of Folies by Contagion.*—The author states that "morbid contagion in mental troubles may be defined as the action of suggestion exercised by a diseased mind upon one or several sane minds, which reproduce it, each according to his individual disposition, either as a simple belief in the disordered idea or in insane manifestation of every form. This state created by contagion has been denominated in the nosological classification of folie, under the name of *folie à deux*, *delire à deux*, etc., expressions which we think too general and which we replace by *folie par contagion*." The author states that this definition has the advantage, first, of comprehending the numerous observations published on this subject, and second of being general; *folie à deux* being only a special effect of suggestion in the morbid psychical sphere; suggestion which acts in so many diverse forms in normal psychical life." I. The article proceeds with an extensive bibliography of the subject, beginning with Baillarger in 1857, who in that year first described *folie à deux*, and mentioning among others Oscar Woods, Jelly, W. Griffin and R. Dewey. II. In order that folie by contagion exist, there must be two principal pathogenic elements: contagion and morbid suggestibility or predisposition. "Morbid contagion is the *raison d'être* of all forms of folie by imposition, of folie by communication, of folie by transmission or by the simultaneous existence of insane phenomena." This was long denied by the authors, as Regis, etc., but is now fully established. Suggestion, the authors say, has three factors: imitative, persuasive and intermental action; this last factor being, according to M. Tarde, the conditions under which one mind is able to act upon another. III. The author reviews the determining causes of the development of folies by contagion. There are three groups of causes; the first accentuates the suggestive elements; the second addresses itself to predisposition, and the third to impression. In the first group are 1st, a long life in common and as intimate as possible; 2nd, habitual ascendancy of the unsound upon him or them of sound mind, etc. In the second group: general wretched condition, illness, intoxications, debilitating vices, etc., physiological states, like puberty, menstruation, pregnancy, accouchment, lactation, menopause. The third group: impressions produced by insane phenomena. IV. In all folie by contagion there are two elements: an active subject, contagious element; a passive subject, element receptive. "The active subject in general exhibits intellectual faculties more vigorous, a will and an energy more powerful than the passive subject: he is, in a word, more suggestive. His intellect, however, may not be superior, may even be inferior to that of the passive subject, the acuteness of the insane phenomena suffices to create the suggestive state. The passive subject is one whose intellectual faculties are enfeebled in various degrees by a predisposition whether hereditary or acquired; in a word, he is suggestible. He may reject the morbid impression when the influence is withdrawn; or he may appropriate, develop

and transform them and give them a form quite personal. The author recognizes three clinical forms of folie: folie imposed, folie simultaneous and folie communicated. The author does not agree with the opinion of Marandon, who thinks that the passive subject never has hallucinations, and that he should not be classed as insane. He cites the cases of a young man and a young woman, in which the latter became the active subject of folie imposed. The young man had cohabited with the woman as his mistress for several years. She developed insanity of the alcoholic type, ideas of persecution, jealousy, hallucinations of hearing and sight, disorders of taste and smell. Her delusions led her to complain to the police, and her jealous threats frightened the young man. He consulted his physician, but under delusion said that the persecutions complained of by the woman were realities. The physician advised that he have the woman taken to an infirmary. He did this a few days thereafter. A month after the woman's admission, he became quite restored to mental health, and the woman, after four months of isolation and treatment, was discharged cured. The difference between folie imposed and folie communicated is that in the former there is merely a transmission of delusions, while in the latter the patient evolves delusions of his own, in addition to those imposed on him. The patients must be separated for their treatment. The author furnishes at considerable length two other cases under his observation, one where the patient was the active agent in the folie by contagion, and the other where the patient was the passive agent. The author concludes that passive subjects of folie imposed must of necessity be considered in a suggestive state and consequently in a medico-legal point of view irresponsible, so long as they are in that state.

2. *On Obsessions and Impulsions (Episodic Syndromes in the Degeneracy)*. Degeneracy betrays itself, says the author, by a certain number of physical signs generally grouped under the domination of stigmata. The external manifestations of morbid syndromes are obsessions, impulsions, inhibitions, perversions of instinct, various delusional troubles, all of which arise from disequilibrium of cerebral departments. Obsession and impulsion have two elements: a center entering suddenly into function, by itself, without being solicited by the need of movement, and a momentary impotence of the will to escape the phenomenon which imposes itself. There is a rupture of the harmony of cerebral centers and exuberance of certain of them. We believe that we may affirm that volitional element has no action in the genesis of obsession. It has been claimed that obsession is a malady of the will. As we see it, says the author, all that occurs, occurs without exercise of the will, and if this last appears to intervene, it is in impulsion only, and in such a case it appears simply suspended, momentarily annihilated as a sequence of the struggle which it opposes against a very superior force, a force due to the *exaggeration* of one or several psychical centers in a state of excitement. The obsedant idea in the degeneracy is born with the individual, remains a long time latent, until the moment when an appreciable cause makes it break forth; then increasing little by little it reaches a maximum and constitutes the morbid troubles called episodic syndromes. The author sets forth with much detail five cases of syndromic states: ideas of homicide, suicide, doubt, theft, perversions, etc. The first was of a single woman, thirty-three years of age, with conscious ideas impelling to homicide, suicide, with fears of all instruments capable of causing death, like knives, needles, pins, etc.; there was perversion of sexual instinct, also onanism. She entered a private hospital, "was submitted to two seances of hypnotism," *suggestion* gave some tranquility, and finally distractions, reading, and above all an alimentation sufficiently abundant, joined to daily moral treatment, improved her state, so that now she has only some abdominal troubles and headache. There were no signs of degeneracy, but clear indications of hysteria. The author remarks that the syndrome of impulsion, contrary to the claims of certain clinicians,

did not lead fatally to action. The second observation is upon the case of a woman of thirty-four, married at the age of nineteen, had 6 children and 2 miscarriages. One son at the age of 16, after typhoid fever, had committed suicide. This misfortune rendered her sad, but under medical care she recovered perfect health. Without any appreciable cause she once attempted suicide by poison, and twice left her home with the idea of suicide; once, thinking of her 5 children, she returned home, and the third time remained in a forest five days without eating, hoping to die by starvation. Admitted to the asylum she said: "I don't know, the idea of suicide came to me suddenly without any reasons. It is stronger than I." In this case the syndrome of suicide was the only one; its access was sudden, and there was no resistance on the patient's part. Such individuals are scarcely responsible for their crime. The third observation was of a woman of forty-one, married. There was a history of gross sexual perversion, onanism. She had impulses to break crockery, to kick at stones in the street, to give blows to persons who were passing beside her. She was for three years a janitress, and believed that in rendering her accounts to the manager she had paid over a sum less than she owed. From that moment she became sad and preoccupied and doubted whether she had not lost some banknotes. In the street she was impelled to rummage the pockets of people, believing that they contained banknotes. Taken to the hospital she saw banknotes everywhere; at sight of notes her face became red, she briskly recoiled and feared to touch them. By degrees, under the moral treatment of the physician, which had great influence with her, she became less anxious and at the end of six months entirely re-established. At the present time she is lucid and touches banknotes without emotion and recognizes the absurdity of her former ideas. In this case there is a co-existence of a certain number of obsessions and impulsions, which appear little by little during a period of five years and ends by a cure, which is the usual termination. Observation four is a case of impulsion to theft and suicide in a woman of forty-five. She was severely ill with typhoid fever, had chorea, hysterical crises. She was employed at a dressmaker's shop; at thirty-seven she took up this work again at Paris, but the handling of the silk enervated her greatly and brought on "crises" almost every week. A second and more severe attack of typhoid rendered her more dull, and at the same time arose the impulsion to theft. The impulsion appeared suddenly and was followed always by the act, resistance almost nil. The object of her theft was always silk, and by preference red silk, and the act was accompanied by sexual orgasm. For a theft at the Bon Marché she was condemned to six months in prison. (A note says that she was reported to have undergone sixteen sentences for numerous thefts in shops.) She was brought to the asylum after having thrown herself from a window on the second floor of the house where she lived; she remained there three months. After an hysterical crisis, she attempted to throw herself before a railroad train, but was prevented by her daughter. The next day, without being completely dressed, she took a cab and went to a shop, where she stole a package of silk. She was arrested and taken to the Infirmary. At the hospital she seems debilitated, rubs her hands constantly, is restless, weeps at intervals. The author considers this an example of the possible co-existence of mental troubles, syndromic and nervous. It shows hereditary degeneracy, re-enforced by fibrile troubles. Observation 5: Case of a married woman, who was brought to St. Anne in 1902 for the third time. Her first morbid episode occurred thirteen years ago, when she had fear of needles, glass and fire. She could not see a candle lighted without fear of seeing it kindle a fire. In buying an object, after paying for it, she doubted she had not paid less than the sum due. She became anxious and frequently wept and was taken to the asylum for the first time. She was discharged after three months completely cured. After an interval of 11 years, she was seized with depression, multiple hallucinations of

hearing, sight, ideas of culpability, of persecution, and some of her former ideas of doubt. She left the asylum in 30 days, again entirely cured. She passed two months in the country, returned to Paris in October, 1901, and by degrees new ideas entered her brain. She became very religious, went to church every day, though before she did not even go on Sunday. She told her husband that she was poisoned, etc. Her troubles became aggravated, and in February, 1902, she was again admitted at St. Anne. Mystic ideas predominated: she complained of suffocation, palpitation, night visions, etc. After three days she became lucid, all had disappeared; she only felt general fatigue. This clinic shows us three forms of trouble; all belong to the same base—degeneracy. There were not three maladies belonging to three periods, for then there would be three different conditions, one attached to each of the morbid periods. The author concludes: "Finally from this exposition of what the clinic has shown us, we think that we should state some particulars concerning a certain number of important syndromes. The group of obsessions and impulsions to homicide and suicide present some special characteristics. The obsessional idea is more tenacious, the struggle on the part of the subject is increased by the will, which opposes itself with a greater energy; in fine, the moral torture reaches a maximum. In what concerns the performance of the act, we should add, and with much justice, that Magnan distinguishes two varieties of impulsions. In the first variety, the onset is sudden, automatic, with no anterior resistance or reflection in the subject (see second case above); in the second variety, the person holds himself for a long time as master of the obsedant idea, or rather at the same instant that he sees himself on the point of yielding, he has recourse to the aid of a member of his family or other such help; 'he gets the start of his environment' (*il prévient son entourage*), according to the expression of Magnan (see second case above). In fine, in the medico-legal point of view persons, who commit crimes under the influence of these morbid impulsions are not responsible, and such persons, as well as the criminal-born of Lombroso, are only, as we have seen, the unbalanced, belonging to the great family of degenerates. The second syndrome, the folie of doubt, demands also some comment, because of the different interpretations which it has received from alienists. Falret, Sr., who first described folie of doubt in an admirable manner, commits the great fault of isolating it too much. He makes of the syndrome a distinct malady of progressive character and a definite prognosis: incurability. Le Grand du Saule, in his monograph, associates it with the delirium of touch; to his mind this intimate association exists of necessity, and the appearance of the delirium of touch marks the debut of a second period in the morbid entity, formed by this association. There are at this time among the clinicians a certain number who still entertain these opinions. As for Magnan and ourselves, we are far from looking upon the folie of doubt under such an aspect; it constitutes merely a syndrome, which may exist in the clinic, either by itself or at times associated with the delirium of touch, and that only by preference, for we may meet it also with other syndromic states. The prognosis is habitually favorable, but on the cardinal condition of never neglecting the patient, of having him each day under one's eye, and of instituting for him a continuous medical and moral treatment. This view is, besides, justified by all our observations. The other syndromes set forth in our cases give a precise idea of the general characteristics indicated at the commencement of this essay. Briefly, in conclusion, we think that we may affirm that this study shows us once more that episodic syndromes, varied as they may be, belong to the same foundation, mental degeneracy; on the other hand, it permits us to place upon the solid foundation of the clinic, in the medico-legal appreciation, faulty and criminal acts committed by individuals belonging to the great class of degenerates."

MR. RICHARDS (Amityville).

ARCHIVES D'ELECTRICITE MEDICALE

(No. 120, 1902, December.)

1. An Adjustable Inductance for High Frequency Resonators. H. GUILLEMINOT.
2. On the Use of an Electrolyte in Parallel to the Primary Current of a Coil for the Production of X Rays and High Frequency Currents. BORDIER and NOGIER.
3. Anesthesia by the High-frequency Brush Discharge in the Ablation of Protruding Hemorrhoids and Hemorrhoidal Condylomata. BILLINKIN.
4. Electric Cerebral Inhibition in Man. STÉPHANE LEDUC.
5. Measuring the Diameters and the Area of the Heart upon the Radioscopic Screen without Drawing. A New Arrangement Adapted to Any Screen. H. GUILLEMINOT.
6. Death and Accidents by Industrial Electric Currents. F. BATTELLI.
7. Congress at Berne. Exhibition of Apparatus for Medical Electricity and for the Production of X Rays.

1. *Inductance for Resonators.*—The author describes his method of more accurately tuning resonators by the introduction of an adjustable self-induction coil in the primary oscillating circuit, a device that was employed by Tesla about 13 years ago.

2. *Electrolytes in the Primary Circuit of Induction Coils.*—The writers recommend shunting the terminals of the primary by a small cell composed of lead plates submerged in very dilute sulphuric acid. Under certain conditions of current the relative spark length of the coil is stated to be considerably increased.

3. *Electrical Anesthesia in the Excision of Hemorrhoids, etc.*—In this paper the author fully describes his mode of using a high-frequency brush discharge applied to the anus to produce anesthesia and constriction of the neighboring blood vessels. With suitable apparatus properly used a painless and practically bloodless operation can be performed.

4. *Cerebral Inhibition.*—A description of a method of producing partial inhibition of the sensori-motor cerebral centers by causing a rapidly interrupted electric current to traverse the brain and spinal cord. The negative electrode is placed upon the forehead and the positive on the lumbar spine. A 50-volt generator of low internal resistance is used to supply the current in conjunction with an adjustable shunt rheostat, while the current is interrupted by means of a Gaiffe mercury interrupter. The sensations of the subjects, Prof. Malherbe and Alfred Rouxeau, upon whom the investigations were made, were as follows: "When the current was at a maximum we could still hear as in a dream what was said; we were conscious of our inability to move and to communicate with our colleagues; we perceived the touches, the pinching, and the pricks upon our arms, but the sensations were dulled like those from a limb fast asleep, etc." No disagreeable after-effect resulted.

5. *Measurement of the Heart Area, etc.*—A short description of the author's new disposition of his previously-described frame with horizontal wires and sliding balls, designed to measure the diameters and the area of the heart, projected orthogonally upon the fluorescent screen. In its new form the frame is placed on the fluorescent side of the screen, and the measurements are read distinctly from the accompanying centimeter scale on glass.

6. *Death by Electric Currents.*—A lengthy and complete paper which should be read in full to be appreciated. The paper is an extensive summary of Prof. Prevost and Dr. Battelli's investigations upon this important subject, and also contains some very interesting remarks upon the electrocution of criminals in this country. Briefly stated, the author finds that low-tension continuous or alternating currents kill by causing cardiac

fibrillation; high tension alternating currents at 1,200 volts or more kill on the contrary by inhibiting the respiratory center. Alternating currents of from 240 volts to 600 volts produce in dogs cardiac fibrillation and often permanent arrest of respiration. High-tension currents, according to the author, do not produce cardiac fibrillation, but possess the property of causing a fibrillating heart to beat coordinately after it has been subjected to a previously applied low tension current. Upon the subject of criminal electrocution the author says: "With the introduction of the new method employed in electrocutions (a high-tension current followed by a low-tension current), the inhibition of the nerve centers is followed by cardiac paralysis. Thus burns are avoided, and the signs of life cease most rapidly. Electrocution, as all other methods of capital punishment, is a disgrace to our civilization, but it is the least repugnant because consciousness is immediately lost and the condemned is not disfigured, nor is the execution bloody."

(No. 121, 1903, January.)

1. Measuring the Actinic Power of the Radiants Employed in Phototherapy. A New Actinometer. BORDIER and NOGIER.
2. On Intra-rachidian Stimulation in Man as a Therapeutic Procedure. J. BERGONIE.
3. On the Maximum Power of Currents Employed in Medicine and the Way to Obtain Them. H. GUILLEMINOT.
4. Electrotherapy and Orthopedics. E. ALBERT-WEIL.
5. The Present State of Electrodiagnostics. LUDWIG MANN.
6. A Five-Cell Electric Bath or Perineal Bath. FELIX ALLARD.

1. *Measurement of Actinic Power in Phototherapy.*—As the authors consider it highly advisable to gain some idea of the intensity of the violet and ultra-violet rays to which the subjects are subjected in phototherapy, they have devised a special form of actinometer by which it is claimed that not only the relative intensity of violet rays can be accurately measured but also the intensity of X rays.

2. *Intra-rachidian Electrical Stimulation.*—A paper describing the effects of faradic excitation of the cord and nerve roots in three patients in whom the lumbar puncture was performed several times. The patients apparently experienced no pain and, luckily, experienced no evil after-effects.

3. *Maximum Intensity of Currents in Medicine.*—A paper based upon mathematical and theoretical considerations from which the author concludes as follows: (1) If a generator with unchangeable constants is employed in conjunction with a variable external resistance, the maximum intensity of current will be obtained by making the external R , equal to the internal r . (2) If the resistance of the external circuit is unvariable and several generators are employed, q elements in parallel must be united into t groups in series, so that $t = \frac{R}{q r}$, in order to obtain the maximum current.

4. *Electrotherapy and Orthopedics.*—This paper is merely a brief sketch written to show that the use of electricity exerts considerable benefit in orthopedics, "but one must not forget that its value depends upon the manner of its application." The writer fears from his experience that the use of the galvanic current in atrophic paralyses in children is inapplicable, owing to the pain produced by the electrodes.

5. *Electrodiagnosis.*—A long summary discussing the merits and demerits of the various commonly employed methods used in electrodiagnosis, which is unsuitable for abstraction.

6. *Electric Perineal Bath.*—A description of an apparatus consisting of five vessels suitably connected to a switch board, so that one of special shape, in which the patient sits, serves as one electrode while the remaining four in which the hands and feet are inserted, form the other electrode.

(No. 122, 1903, February.)

1. On the Electric Treatment of Basedow's Disease. TH. GUILLOZ.
2. How to Understand the Contra-indications to the Electric Treatment of Uterine Fibromyomata. M. LAQUERRIÈRE.
3. The Treatment of Gonorrheal Arthritis in the Acute Inflammatory Stage by Strong Continuous Currents. LOUIS DELHERM.
4. The Action of Ozone Upon the Bacillus and the Toxine of Diphtheria. F. ARLVING and M. TRONDE.

1. *Treatment of Basedow's Disease.* The author reports two cases of Basedow's disease considerably ameliorated by the electro-cataphoretic method of treatment, in which iodine was introduced into the tissues by a strong continuous current.

2. *Treatment of Fibromyoma.* After discussing the various varieties of electric treatment and specifying those forms of this trouble to which the different varieties of current are most applicable, the author gives a number of absolute and relative contra-indications which should be read in the original to be appreciated.

3. *Treatment of Gonorrheal Arthritis by the Continuous Current.* The author advises that in cases of hyarthrosis, or gonorrheal arthritis a galvanic current of from 20 to 60 m. a. be applied as soon as possible once or twice a day. Large kaolin electrodes are to be used.

4. *Action of Ozone on the Diphtheria Bacillus.*—These authors find that by exposing cultures of Loeffler's bacillus to ozonized air (.025 gram to 1 liter the effect on the bacilli is as follows: (1) Ozone cannot kill the bacilli in a nutritive liquid. (2) It can attenuate their growth for 48 hours, although the proliferation after this period is less marked than in ordinary cultures. (3) It diminishes the virulence of the bacilli so that inoculated guinea pigs survive, and in those that die only lesions at site of the inoculation are found. When the toxin was exposed to 200 liters of the above-mentioned ozone mixture, the animals survived a 1 cc. injection, although the control animals died in 24 hours from a dose of 0.5 cc.

R. H. CUNNINGHAM.

JOURNAL DE NEUROLOGIE

(Vol. 7, 1902, No. 21, November 5.)

1. Sensorial Excitations as a Cause of Epileptic Seizures. CH. FÉRÉ.
2. Occipito-Cerebellar Syndrome. L. MARCHAND.
3. Exophthalmic Goiter. Treatment by Thymus. A. BIENFAIT.

1. *Sensory Excitation in Epilepsy.*—The author emphasizes the fact that sensorial as well as sensory excitations may provoke epileptic attacks, and relates the following cases: (1) In an idiot of eighteen years, subject to both diurnal and nocturnal attacks of grand mal, the diurnal seizures were found constantly to follow sensorial excitations. Sudden noises, sudden variations in intensity of light, a current of air, and even a strong odor sufficed to bring on a fit. (2) In a man of sixty-five years of age, epileptic from his youth, and having two or three fits monthly, loud noises and sudden changes in light intensity would often bring on an attack. The author saw a typical grand mal seizure produced in this case by a sudden blow on the table behind the patient. The lesson taught is that in epileptics careful investigation into the exciting causes of their attacks should be made, and attention should be paid to any idiosyncrasies in this particular which they may present.

2. *Occipito-cerebellar Syndrome.*—The author describes the case of a woman of forty-two years of age, who had had occasional attacks of epilepsy for five years. Seized suddenly with headache, diarrhea and vomiting, she speedily developed loss of vision, going on to nearly complete blindness, staggering gait, Romberg's symptom and exaggerated reflexes.

The ophthalmoscopic examination showed double pupillary stasis. She also had several fits. After eight days the gastrointestinal symptoms improved, but the next day she went into status epilepticus. Learning at this time that her husband had been syphilitic, the author began intramuscular injections of biniodide of mercury and iodide of potassium. She commenced to improve, and in about two months had regained her vision, and the headache and motor symptoms had disappeared, though the epileptiform attacks persisted. The author thinks that there is no doubt about the syphilitic origin of the symptoms, and diagnoses a gumma at about the insertion of the falx cerebri into the tentorium.

3. *Exophthalmic Goiter, Treatment by Thymus*.—The author discusses the pathogeny of Basedow's disease, and narrates some experiments of his own upon rabbits, in which incisions into the medulla about and into the restiform bodies produced some of the symptoms of this disease. That these symptoms probably arise from this region he thinks is shown by the results of experiment and by the finding there of lesions in a certain number of cases of Basedow's disease. Certain other maladies, notably tabes, which are progressive, have occasionally been found to cause some of the symptoms of Basedow's disease when they extend to the medulla. These symptoms, again, may arise reflexly or be neurotic in origin; and, lastly, the center in question may be susceptible to the action of certain poisons. The author thinks that the thyroid and thymus glands may in some way be complimentary in action, hence suggests the use of extract of the latter, and gives a report of a case in which he used this remedy with some, though not very positive benefit.

(Vol. 7, 1902, No. 22, November 20.)

I. Dementia Præcox. F. MEEUS.

1. Upon the basis of his observations in 47 cases, the author discusses the etiology of, and attempts to construct a symptomatology for, dementia præcox. The disease, he thinks, is not limited to early life, but may arise at a later period, and should be considered as a special form of mental trouble, with characteristic mental and motor symptoms, proceeding certainly to dementia. The characteristic negativism, stereotypy, etc., he regards as due to disturbance of association, which prevents regular and orderly discharge of impulses, resulting in inhibition, broken by sudden and irregular or ill-timed explosions. The term dementia præcox he regards as too indefinite, and suggests that it be replaced by that of "hebephreno-catatonic dementia."

(Vol. 7, 1902, No. 23, December 5.)

1. Theory of Reflexes Cutaneous and Tendinous. C. HELDENBERGH.

2. Congenital Facial Diplegia. O. DECROLY.

1. *Reflexes*.—A discussion of the physiology and pathology of reflexes, which embodies no original investigations, and does not lend itself readily to review.

2. *Congenital Facial Diplegia with Ocular Paralysis and Trouble with Deglutition*.—Report of a case of this kind in an infant of seven weeks old. The author is inclined to think it due to a lesion of the bulbar nuclei.

(Vol. 7, 1902, No. 24, December 20.)

1. The Intracellular Network of Golgi in Nerve Cells. S. SOUKHANOFF.

2. Chronic Poliomyelitis. DE BRAY.

1. *Intercellular Network*.—A review of the findings of Golgi and Veratti, with a description of the intracellular network as demonstrated by the author himself, especially in the cells of the sympathetic ganglia, by the method of chrome-silver impregnation of Veratti. The author does not regard this intercellular network as conductive in function, but rather as analogous to the intracellular canaliculi.

2. *Chronic Poliomyelitis*.—The author describes the case of a man sixty years old, who subsequent to a fall developed atrophy of the hand

muscles of the Duchenne-Aran type, with some kyphosis in the upper dorsal region. From a consideration of the situation of the special nuclei of the hand muscles, in the lateral region of the anterior horn, and their blood supply differing from that of the other cells of the region, he draws the inference that in this case there was a vascular lesion produced at the time of the injury, and the cells of the nucleus in question atrophied in consequence, while the other cell groups in that part of the cord escaped.

ALLEN (Trenton).

NEUROLOGISCHE CENTRALBLATT

(Vol. 22, 1903, No. 1, January 1.)

1. A Contribution to the Feeling of Consciousness. A. PICK.
2. Concerning Hysterical Confusion and Mental Aphasia. A. WESTPHAL.
3. Concerning a Developmental Defect of the Nose (An Undescribed Stigma of Degeneration). H. GUDDEN.

1. *Feeling of Consciousness.*—Pick considers in this paper the *bekanntheits Gefühl* as a condition in hysteria, sexual neurasthenia, epilepsy, etc., and discusses the psychology of the conditions.

2. *Hysterical Confusion.*—Westphal reports a case of hysteria with a confusional condition in which the patient cannot answer questions, and when she did answer them the simplest questions were wrongly interpreted or wrong answers were given. The question as to whether true clonus is present in hysteria, is, according to the author, determined positively in this case.

3. *Nasal Stigma of Degeneration.*—Gudden pictures a patient with a defective development of the cartilage and tissue of the *alæ* of the nose and considers it a stigma of degeneration.

Vol. 22, No. 2, 1903, January 16.)

1. Concerning Disturbances of Sideward Movements (Flank Gait) in Hemiplegics. v. SCHÜLLER.
2. Hedonal and the Nervous System. v. LAMPSAKOW.
3. Autogenous Nerve Regeneration. A. BETHE.
4. Answer to No. 3. E. MÜNZER.
5. Hysterical Confusion and Mental Aphasia. A. WESTPHAL.

1. *Concerning Disturbances of Sideward Movements (Flank Gait) in Hemiplegics.*—The author says this is a new differential symptom for the diagnosis of organic hemiplegia from functional hemiplegia. It depends on the fact that a hemiplegic patient can walk sideways towards his palsied side with little or no difficulty, but on account of the spasticity and lengthening of the palsied leg, he has considerable difficulty in walking sidewise in the opposite direction, and must needs drag the paralyzed leg.

2. *Concerning the Action of Hedonal upon the Animal Economy.*—V. Lampsakow gives the results of his psychological study of a new hypnotic, hedonal. He finds that it is an active and harmless hypnotic: much stronger than urethan, without any disturbance of the heart or respiratory organs; that it can be given preparatory to the administration of chloroform to facilitate narcosis; that it is more valuable as a hypnotic in anemic weak patients than chloral, and that it may be given by mouth or rectum, but cannot be administered hypodermically.

3. *Autogenous Nerve Regeneration.*—A. Bethe presents a controversial article with Münzer concerning statements of the latter in a previous article in this *Centralblatt*.

4. *Answer to No. 3 (Bethe) by E. Münzer.*

5. *Concerning Hysterical Confusion and Mental Aphasia.*—Westphal renders a medico-legal contribution to disturbances of memory in functional conditions and malingering of mental disturbances.

MCCARTHY (Philadelphia).

ALLGEMEINE ZEITSCHRIFT FÜR PSYCHIATRIE.

(Vol. LX, 1903, Heft. 1 and 2.)

1. On Ganser's Symptom. LÜCKE.
2. Induced Insanity. F. WITTE.
3. Cases of Simulation. BOLTE.
4. Graudenz Penal Institution. SANDE.
5. Contribution to Study of Paranoia. H. SCHNEIDER.
6. Conception of Hypochondria. A. PICK.
7. Exhibitionists before the Law. G. BURGL.
8. Asylum at Strelitz. SERGER.

1. *On Ganser's Symptom.* Although not previously unknown, Ganser in 1897, called special attention to a peculiar condition in hysterical subjects, characterized especially by confusion, childish bearing and an apparent inability to answer simple questions as to names, dates, and other ordinary matters, or to recognize persons or objects formerly well known. Common among those who have come into conflict with the law, the conduct of these persons under examination may readily arouse a suspicion of simulation. In consequence of the presence of other definite symptoms of hysteria, however, Ganser thinks the manifestation a genuine symptom-complex, and in this he is supported by other observers. The author discusses this symptom and gives the histories of four illustrative cases. The matter he considers important on account of its medico-legal bearing, as the symptom is especially common in prisoners with hysterical mental disturbances. The diagnosis between this symptom and simulation upon the part of the patient, is to be made upon the presence of hysterical stigmata, periodical attacks of anxiety, confusing of persons, and terrifying hallucinations, together with a characteristic stupid and at the same time tense expression of countenance, and generally confused appearance of the patient. The symptom has been described by Nissl as occurring in katonias, but the author regards it as being due in these cases to a hysterical groundwork.

2. *Induced Insanity.*—The histories of two cases, one that of a man of 59 years, presenting a typical example of paranoia with expansive and persecutory delusions, the other that of a woman of 51 years, of neuropathic constitution and heredity, and addicted to the abuse of alcohol, the housekeeper of the first patient. The second patient, in consequence of the troubles of her employer, being left without resources, began to drink more heavily than usual, and developed a psychosis of strongly alcoholic character, with hallucinations and delusions of persecution. Interned in the same asylum as her employer, the picture changed to one of paranoia with expansive and persecutory delusions, almost identical in character with those of the former. The author thinks that the influence of the stronger personality of the man, acting for years upon that of the weaker and less educated woman, already predisposed through neuropathic constitution and alcoholic indulgence, is responsible for the clinical developments in the case.

3. *Some Cases of Simulation.*—Clinical histories of six cases of this character. Not suitable for abstraction.

4. *The New Insane Department of the Graudenz Penal Institution.*—Not suitable for abstraction.

5. *A Contribution to the Study of Paranoia.*—A clinical history extending over the thirty-six years of life of a typical paranoiac, with a lengthy discussion.

6. *On the Conception of Hypochondria.*—The author thinks that hypochondriacal delusions are often built up upon a material basis. To illustrate his views he gives histories of the cases of two middle aged women in each of whom gonorrheal trouble of the genital organs led to depression with persistent hypochondriacal delusions. One case was improved by local

treatment, but in the other the morbid ideas persisted in spite of subsidence of the genital disease.

7. *Exhibitionists before the Law*.—As adviser to the court the author has had occasion within four years to examine twenty-five persons accused of exposing their genital organs in public. He thinks that in the great majority of cases this act is a manifestation of mental disorder. Each case he thinks should be considered under the following heads: (1) Is the act due to mental weakness? (2) To uncontrollable impulse? (3) Is it the result of an imperative conception ("Zwangshandlung")? (4) An accidental occurrence? (5) Due to carelessness? (6) Is it the voluntary act of a mentally normal individual?

Under 1 he includes both congenital and acquired mental weakness. In these cases the act may be due to lack of ethical feeling, and of inhibitory contra-conceptions. It is observed in imbeciles of various grades, in paralytic and senile dementia, in epileptic delirium, after trauma, and in the pathological reaction to alcohol common in epileptics and neurotic subjects.

(2) The cases due to uncontrollable impulse may result from sudden vivid hallucinations or delusions, and sometimes coming on from time to time appear to be a manifestation of periodical insanity ("periodical sexual psychopathy").

(3) The cases in which the act is the result of imperative conceptions, the author separates from those due to impulse. The presence of an imperative conception alone does not necessarily make the patient legally irresponsible, but it may be accompanied by an effort of such intensity as to cause an obnubilation of consciousness, in which case self-control is lost and mental irresponsibility may ensue.

(4) Exhibition may occur accidentally through forgetfulness about adjusting their clothing upon the part of weak-minded or mentally confused individuals.

(5) Especially in old and feeble-minded persons there may be such carelessness as urinating or adjusting a truss in the street.

(6) Lastly, exhibition may be deliberately practiced, generally under great sexual excitement, by mentally normal individuals, usually upon meeting one of the opposite sex in a lonely place. In each case an inquiry into all the circumstances of the case attending the misdeed should be made, and if possible the statement of the patient in his own language should be secured. By so doing the physician will often be able to detect gaps in the memory which indicate the presence at the time of some disturbance of consciousness (epilepsy?). Each case should be judged upon its merits. A history of repeated exhibitions, especially in a very public place before numerous witnesses, points almost unerringly to mental aberration.

8. *The Asylum at Strelitz*.—Description of a newly erected building for 180 patients. Unsuitable for abstraction.

ALLEN (Trenton).

Book Reviews.

INTERNAL SECRETIONS AND THE PRINCIPLES OF MEDICINE. By CHARLES E. DE M. SAJOURS, M.D., Vol. I, Illustrated. F. A. Davis Co., 1903.

The first volume of this remarkable work contains eight hundred pages almost exclusively solid reading matter, the illustrations being chiefly upon plates. It is an attempt to arrive at new theories and principles regarding certain obscure points in physiology, by a careful study of the observations and experiments of others. While it has generally been the practice of the world to accord the highest praise to the original investigator, it must not be forgotten that the man who organizes the mass of facts collected by others, puts them in an available form, and deduces from them new ideas regarding their essential relations, may contribute more to the actual advancement of knowledge than the accumulator. It must not be forgotten that the physicist, Young, takes his rank among the world's great men although his actual laboratory work in physics was comparatively insignificant; that Herbert Spencer has done much to mould English thought in biology, although he was not a great original investigator. To those who can accomplish both—that is, collect the facts and analyze them, as did Darwin—highest rank in science is ever accorded. Dr. Sajous is the analyst. He has made, he believes, some valuable discoveries regarding the rôle of the adrenals and the pituitary body. Briefly stated, his theory is that the adrenal secretion poured into the blood endows the hemoglobin and the plasma with an affinity for oxygen, that the plasma carrying the oxygen then circulates among the tissues and accomplishes the internal respiration. This function gives the internal secretion of the adrenals an overwhelming importance. The adrenal glands are controlled by the anterior portion of the pituitary body, and the functional efficiency of this is in turn sustained by the secretion of the thyroid gland.

These views are sufficiently novel; the question arises: What claim have they to our credence? The first chapter deals with the physiology of the adrenal glands. To us the conclusions in many cases appear to be strained. The proof offered is inadequate. The experimental material is obviously carefully selected, but even then does not suffice to confirm the deductions. Take, for example, the section on the physiological effects of poisons, in which the facts are stated by the investigators entirely without reference to the adrenal glands, and yet changes are explained by Sajous with reference to these organs. Take, for example, the relation of the adrenal glands to chlorosis: the conclusions must appear to any clinician to be forced. Of course the reviewer must guard against too general condemnation of any strikingly revolutionary hypothesis, and it is impossible to give an adequate analysis of the book in the brief space allotted to this review. There is one point, however, to which we feel that attention should be called, that is the statement that the posterior pituitary body is the chief center of the nervous system to which the afferent impulses go and from which the efferent impulses arise, so that it practically governs the brain. To one who has studied the histology of this organ it seems incredible that a mass so scantily furnished with cells or nerve fibers could possibly accomplish all that is ascribed to it. If its embryology is studied it appears more than reasonable to assume that it, like the pineal gland, is a relic of a former organ, and comparative anatomy lends color to this view. What then, has Dr. Sajous to offer against it? Very little, indeed: Its position, which we admit is central enough; the fact that injections of its substance are said to increase the blood pressure; and

the fact that Berkeley's investigations have shown that it actually does contain parts of neurones.

Perhaps we have been rather harsh with this book, but we feel that Dr. Sajous has not made out an entirely good case for the plaintiff. Nevertheless, it is well written, indicates a vast amount of industry, and is exceedingly suggestive. The publishers have done their part well.

SAILER.

A SYSTEM OF PHYSIOLOGIC THERAPEUTICS. Edited by SOLOMON SOLIS. COHEN, A.M., M.D., Senior Assistant Professor of Clinical Medicine in Jefferson Medical College, etc. VOL. V, PROPHYLAXIS, PERSONAL HYGIENE, CIVIC HYGIENE, CARE OF THE SICK, by JOSEPH MCFARLAND, M.D., Professor of Pathology, Medico-Chirurgical College, Philadelphia; HENRY LEFFMANN, M.D., Professor of Chemistry in the Woman's Medical College, Philadelphia; ALBERT ABRAMS, A.M., M.D., formerly Professor of Pathology Cooper Medical College, San Francisco, and W. WAYNE BABCOCK, M.D., Lecturer on Pathology and Bacteriology, Medico-Chirurgical College, Philadelphia. Illustrated. VOL. VI, DIETOTHERAPY AND FOOD IN HEALTH, by NATHAN S. DAVIS, JR., A.M., M.D., Professor of the Principles and Practice of Medicine in Northwestern University Medical School; Physician to Mercy Hospital and Wesley Hospital, Chicago. Philadelphia, P. Blakiston's Son & Company.

In the preface of Volume V of this series we read, "The following pages contain an epitome of what is essentially the natural history of medicine." Such is the broad scope adopted by the editor, who takes a special pleasure in this volume "because of his success in having the subject of prophylaxis brought into close relation with that of treatment at so many points. The artificial conditions of civilization, the diversity, complexity and strenuousness of the activities of modern life, the reciprocal influence of individuals upon communities and of communities upon individuals render it necessary in a work of this kind to consider problems in economics, engineering, manufacturing, architecture, pedagogics, commercial intercourse, taxation, municipal government, etc. These have been brought together and studied in their bearing upon the subject and on each other, and are placed before the reader in as concise form as would seem possible. The bacteriology of the infectious diseases, the distribution of animal parasites, and the whole subject of the spread of disease by insects and other agencies form very important portions of the book. The care of the sick-room is discussed from the physician's point of view as a worthy department of therapeutics. The recent theories of immunity, artificial defences against disease, hygiene in cities, disposal of waste, disposal of the dead, hygiene of women at special periods, care of patient, and special nursing are headings which indicate some of the contents; but in a limited review of this many-sided treatise it is impossible to dwell upon the subject-matter in any details. Suffice it to say that we believe this to be one of those books which belong in the library of every physician.

In Volume VI we have a treatise on Foods, and as this subject has been dealt with by numerous authors, this addition to the list demands less notice from the reviewer than does the preceding volume of the series. The consideration of diet is not merely a question of how to feed the sick, but also of what is proper feeding for those who are in health; and this prophylactic phase of the subject is not forgotten by the author. In a food the chemical composition is important, but it does not indicate the suitability or unsuitability of that food for human consumption. The author, therefore, after describing the chemical nature of the fundamental food-bodies and their transformation in digestion, proceeds to determine which of these enter into the composition of the various food-stuffs (eggs, cereals, meat, etc.), and by what means and to what extent food-

stuffs are rendered wholesome. First the diet in health at different periods of life, infant feeding, the diet of brain-workers, and the ill effects of food are given detailed treatment; then follow chapters on the feeding of the sick in the different infectious diseases, diseases of the blood, kidneys, digestion, circulation, etc. A chapter is devoted to feeding in nervous diseases, and includes diet-tables for neurasthenia and epilepsy. Undoubtedly the book is a mine of information about all the ordinary foods and their modifications, with their uses in health and disease, and even the subject of rectal feeding is not neglected. Each of the volumes in this series so far published is authoritative in its particular field, and the editor is to be congratulated on his choice of contributors.

W. A. BASTEDO.

MEDICAL MICROSCOPY, designed for students in laboratory work and for practitioners, by T. E. OERTEL, M.D., Professor of Histology, Pathology, Bacteriology and Clinical Microscopy, Medical Department, University of Georgia. With 131 illustrations. Philadelphia. P. Blakiston's Son & Co. 1902.

Microscopy is a new and growing science in which those who graduated more than a decade ago received for the most part but scanty instruction. But the more complete works on the subject offer a bewildering mass of material from which it is difficult for the unskilled to cull what will be of service to him. This has been kept in mind in the compilation of this treatise, and where possible the simpler processes have been chosen, and usually but one method given whereby to reach a certain result. The choice of material has been excellent, sufficient details being included to enable even a novice to fix, section and stain tissues, to prepare bacterial cultures or slides, to stain the blood or count its corpuscles, to determine the tubercle or other bacilli in the sputum, and to appreciate the significance of sediments in urine. The saliva, stomach contents, feces, vaginal secretions, etc., are not dwelt upon at length. The many illustrations, some of which are colored, are an important addition to the text; a few of the micro-photographs, however, would be unrecognizable without their labels. We can recommend this book as a practical guide not only to the uninstructed but also to those who have had their course in microscopical studies. W. A. BASTEDO.—

News and Notes.

THE STEVENS' TRIENNIAL PRIZE of Columbia University for original research was awarded to Doctors L. Pierce Clark and Thomas P. Prout of New York upon "Status Epilepticus: A Clinical and Pathological Study of Epilepsy."

A USEFUL WORK on diagnosis—Hill's Reference Chart, noted in our advertising pages, is a very handy little volume on diseases of the nervous system and of the muscles. It will be found particularly serviceable to those interested in nervous diseases. Write to the publishers for a copy. See page xxix of our advertising forms.

PATHOLOGIST AT CRAIG COLONY.—Dr. B. Onuf has been appointed resident pathologist at the Craig Colony for Epileptics at Sonyea, N. Y.

Dr. Onuf is a graduate of Zurich, Switzerland. He acted for two years as assistant to Prof. Forel at the Insane Hospital Burghoelzli at Zurich, afterwards devoting eight months to the study of ophthalmology with Prof. Haab in Zurich. Later he located in Brooklyn, N. Y., where he began to devote himself to neurology. He was affiliated with Dr. Sachs' clinic in New York for a number of years, and later with the nervous clinic at Long Island College, where he gave clinical and didactic instruction to the students. He served as neurologist to St. Catherine's Hospital and consulting neurologist of the dispensary of the Jewish Hospital in Brooklyn. From 1896 to 1902 he was associate in pathology at the Pathological Institute of the New York State Hospitals.

Among his scientific contributions may be mentioned a monograph written in conjunction with Dr. Joseph Collins, entitled "Experimental Researches on the Central Localization of the Sympathetic with a Critical Review of its Anatomy and Physiology."

In selecting a pathologist for the colony, the chief aim was to utilize the unusual opportunities given at the colony for studying the causes of epilepsy. The work is to be not only pathological but also clinical, and it is from this combination that fruitful results are hoped for.

RECENT PROGRESS IN MATTERS PERTAINING TO THE CARE OF THE INSANE IN THE STATE OF NEW YORK.—Some of the measures of improvement in the care of the insane of the State of New York carried through during the past few months by the Lunacy Commission are as follows:

1. The Pathological Institute has been reorganized and more than sixty of the medical men connected with the staffs of the fourteen State hospitals have been instructed during the past winter at the Institute on Ward's Island in the recent development of psychiatry along clinical, pathological, psychological and clinical lines. The legislative appropriation for the Institute is now \$25,000 annually.

2. The hospitals have been opened to medical internes in the same manner as general hospitals. Last year sixteen clinical assistants entered the service in this way, and this year the number is nearer thirty.

3. The legislature recently passed the Lunacy Commission's bill for the appointment of a medical inspector to ensure a more thorough inspection of the thirty-nine institutions under its charge, viz.: 23 private retreats, 2 criminal asylums, and 14 State hospitals for the insane. Such inspection, especially of the private asylums, in which there are about 1,000 patients, has never been adequate.

4. To remedy overcrowding the Lunacy Commission proposes to construct a new hospital in the territory north of Albany and Troy on the colony system—a scheme similar to that of the Craig Colony for epileptics will be carried out. The site will be selected and plans made this year.

This colony for 1,500 to 2,000 patients should be ready inside of three years.

5. Three tuberculosis hospitals, each with a hundred beds, will be constructed this summer at a cost of \$90,000 at Middletown, Utica and Binghamton, on the grounds of the State hospitals located there; and the plans made by Dr. Peterson and the State architect embody the main features of such hospitals described in the King Edward Prize Essays. In the meantime tent life for the tuberculous insane has been in vogue at the Manhattan State Hospital East (under Dr. Macdonald) for two or three years and for a shorter time at Binghamton and other of the State hospitals.

6. The country colony for a few of the working classes of the insane, as an offshoot of the Utica State Hospital, has been enlarged. A similar colony has been established at the Willard State Hospital, and two are in existence at the State Hospitals at Binghamton and Poughkeepsie.

7. A new departure this year is the creation of a summer camp for between 40 and 60 insane on the lake shore, about fifteen miles from the Rochester State Hospital, which is now in operation to the great delight of both patients and attendants.

8. The feature of nurses' homes having been found so useful at some of the hospitals two additional ones will be put up this summer, one at Kings Park State Hospital, Long Island, for 300 nurses, and one at the Gowanda State Hospital for 100.

9. Six or seven residences for superintendents and separate houses for the medical staffs will be put up this season at as many of the State hospitals, thus removing the officials from the central main buildings and utilizing the vacated space for patients.

10. A bill providing for emergency commitments, recommended by the Lunacy Commission, was passed by the legislature at the last session. Copies of this law have been sent to all examiners in lunacy of the State. It is believed that this will mean great good to the insane and prevent the all too frequent incarceration of urgent cases in jails and station houses.

11. The improved ration brought about during the past six or eight months, though entailing an additional cost to the State of a hundred thousand dollars per year or more, has added greatly to the comfort of the patients and to the satisfaction of the medical officials and various visiting boards.

12. A strong effort is being made by the Lunacy Commission to increase the number of deportations of alien insane, and through the efforts of New York State the federal government passed a law making the limit three years instead of one; i. e., an immigrant becoming insane within three years after landing in the United States may be returned to his own country.

13. The movement for the establishment of reception hospitals for acute curable cases in the large cities has gained strength. While the bill for the psychopathic hospital for New York city, which was to be the first of several such reception hospitals, failed to pass this year, it is believed that success will attend the Commission's efforts during the coming session.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

MYOCLONUS MULTIPLEX AND THE MYOCLONIAS; REPORT
OF CASES AND AN ATTEMPT AT CLASSIFICATION.

BY CHARLES L. DANA, M.D.,

PROFESSOR OF NERVOUS DISEASES, CORNELL UNIVERSITY MEDICAL COLLEGE.

I. N.

Myoclonia is a general term covering all the diseases in which myoclonus or muscle-twitching is a prominent symptom. This is the definition of Foster's Dictionary, and the use of the word in this general sense is the proper one. It has, however, been used by Seeligmüller (1887) to indicate the same thing as paramyoclonus multiplex, and Unverricht used it (1891) in about the same way.

Friedreich reported his case of paramyoclonus multiplex in *Virchow's Archivs*, Vol. 86, 1891. During the ten years following a number of cases which were more or less like his were put on record. When Unverricht published his "Myoclonia" (1891), he recognized that his cases were different from those of Friedreich, and he separated them into a group, called the "familial myoclonias." Following Unverricht, there came reports of a number of cases of myoclonia, some of which resembled this "familial" type, and some, more or less (rather less than more), the original case of Friedreich. The history of these is given in the monograph of Lundborg: "Ueber Degeneration und degenerierte Geschlechter in Schweden," published in 1891, and the more recent monograph of Clark and Prout, *American Journal*

of *Insanity*, No. 2, 1902. The criticisms upon these various papers indicated different views. Thus Wollenberg and Boettiger concluded that the Friedreich type of case belonged to hysteria, and that there really was no such independent disorder as paramyoclonus. The "familial" type of Unverricht was thought to belong to the "degenerative chorea" of Huntington. In this view Moebius coincided. Schultz, on the other hand, placed the Friedreich type amongst the forms of convulsive tic and the Unverricht type amongst the degenerative choreas. Marie was of the opinion that paramyoclonus is a special disorder, and with this view Oppenheim, Bechterew and Strümpell appear to agree.

The view which I entertain differs somewhat from any of those expressed heretofore, though it is most in harmony with that of Schultz. It seems to me that the cases which were most in harmony with Friedreich's original case, belong to the group of myokymias, or neuro-muscular spasms without special locomotor effect, and are spasms dependent upon a disturbance of the peripheral motor neurones. There are certainly groups of cases of this kind, as is shown by those recently reported by Hunt¹. Most of the other cases reported as paramyoclonus belong to the category of hysteria, or functional neuroses, or to convulsive tics. The "familial group" of myoclonias described by Unverricht and Lundborg seem to me to belong certainly to the degenerative choreas, though they are pretty widely separated, as a rule, from the ordinary group of Huntington's chorea, being much more severe in their manifestations, and rapid in their course.

A further discussion of the reasons for this grouping will be given later.

The following cases illustrate four different groups or types of myoclonia.

1. Myoclonia of the spinal and peripheral type, including myokymia, fibrillary myoclonus of Kny, and Friedreich's paramyoclonus multiplex.
2. Myoclonia of functional and hysterical type.
3. Myoclonia of the convulsive tic type.
4. Myoclonia of degenerative chorea and epilepsy. (Myoclonus-epilepsy, myoclonia of family type.)

¹Hunt: JOURNAL OF NERVOUS AND MENTAL DISEASE, June, 1903.

Personal Cases. Case I.—*Myokymia and myoclonus (paramyoclonus of Friedreich).* The following case showed very typically the phenomena of myokymia, with at times myoclonia and myotonic contractions with locomotor effect, irregularly distributed and involving muscles not under voluntary control. It is a case resembling in many ways that of Friedreich's, though the dominant feature was the myokymia, and resembles in this more the fibrillary myoclonus of Kny.

Summary.—Male, 23, workman, family and personal history negative. At 22 had twitchings of muscles, and painful spasms of legs and arms. Improved, but twitchings never entirely left him; cramps ceased recently. Entered hospital for general weakness, and diffuse pains in bones of legs, pelvis and head. No objective signs of any organic disease. Muscles of all parts of body in continual play of fascicular and fibrillary spasm, causing irregular tremor in fingers and toes, and sometimes locomotor effect of finger and leg. Fascicular spasm in thighs increases and culminates in myoclonus and myotonus of one or both hamstring muscles. Similar myoclonus in biceps. No painful spasm now.

Male, aged twenty-three years, single, Slav, was brought to Bellevue Hospital, complaining of pains in the legs and arms, and great general weakness. He stated that one year ago he had had a severe attack of pains which were called rheumatic. He at that time had also muscular contractions such as were still present and had had at times "cramp-like" contractions of the muscles of the legs and arms. He improved, but continued to have at times cramps, pain, and muscular twitchings. Of late this condition had grown worse. The patient was brought to the hospital, complaining of weakness and pains in the legs, thighs and head. He was a rather small man, somewhat emaciated, but with fairly good muscular development. His body was covered with a fine eruption which was found to be due to scabies.

As the patient lay in bed fine fibrillary spasms could be seen playing over nearly all the muscles of the body. They were very exquisitely marked in the face, where they affected especially the small muscles, such as the buccal, risorius, and nasal. The contractions were not very frequent, not more than one or two being seen at a time, and they occurred in single "flashes," jumping from one spot to another, they affected the two sides about equally. No general motor effect resulted. Similar phenomena

occurred in the forearms and in the intrinsic muscles of the hands, the lumbricals and interossei. Here they recurred frequently, causing slight locomotor effect and a condition like a fine tremor, only the fingers were also irregularly flexed, abducted and adducted. When the long flexors were involved the fingers were rhythmically flexed a trifle, the movement sometimes running into a tonus with contraction of the fingers. The movements occurred in the forearms, arms, shoulders, pectoral, abdominal and posterior trunk muscles; here they were more violent, but were less frequent and caused no locomotor effect, except that occasionally the biceps would move the forearm. The calves and intrinsic muscles of the toes were affected just as were the upper extremities. On making the patient lie on his face the movements could be seen most strikingly of all in the muscles of the calf, posterior part of the thigh, and in the buttocks. Here then were frequent fibrillary contractions and also wave-like contractions running along the muscle from center to periphery. On flexing the leg at right angles to the thigh, the inner hamstring muscles would begin to contract more vigorously and finally ended in a distinct myoclonus or more often in a tonic contraction which held the leg flexed for several seconds. The same thing occurred in the outer hamstring muscles and sometimes in both, holding the leg in flexion. Thus there were here fibrillary contractions, wave-contractions, finally ending in a distinct clonic or tonic contraction of the whole muscle, sometimes with locomotor effect. The phenomenon occurred in muscles such as the inner hamstring, not under direct voluntary control. The contractions could be brought out by manipulating the leg, and by blows of the hammer. They were unaccompanied with pain, and the flickering spasms occurred at times 40 to 60 times a minute. The myoclonus and myotonus were observed only when the leg was flexed and the muscles relaxed. A similar condition but much less marked was present in the biceps. The myokymia continued during sleep. There was no atrophy, contracture or paralysis. The general muscular irritability was much increased and immense myoid tremors could everywhere be easily produced. The electric reactions were not tested. The muscular power was fairly good. The deep and skin reflexes were normal. There was no sensory disturbance of any kind. The special senses were normal. He had no stigmata of hysteria. The patient complained of general pains, which seemed to be mainly in the bones. There was no tenderness or swelling of joints or muscles. His mind at first seemed at times confused. He presented no objective symptoms of any visual disorder, or of disease of the central nervous system. The blood and urine were normal, eosinophiles normal. He could walk, but his weakness kept him in bed. He improved

and the eruption grew less under mercurial treatment, but the muscular phenomena were unchanged.

Taking the whole history of the case it will be seen that he had painful cramps as in Friedreich's case, clonic twitchings of the muscles of the thighs and arms, not of the peripheral segments, muscular contractions of the whole body of a muscle, sometimes with and sometimes without locomotor effect, and that muscles were affected not under voluntary control. In all these respects, the phenomena were like those of Friedreich's case, but in addition he had a widespread and remarkable myokymia, involving nearly all the muscles. The spasms could not have been of cerebral origin, certainly not cortical, and it seems to me that the seat of the trouble must be either in the spinal cord or peripheral motor neurone,—which is the pathology attributed by Friedreich to his case.

It shows a close relation between ordinary myokymia and the severer types of bundle clonus, to which the term paramyoclonus multiplex, if used at all, should be applied.

Case 2.—*Paroxysmal myoclonus of functional (hysterical and family type. Summary.*—Male, aged forty-two; mother suffered from similar attacks. Healthy till the age of eleven years, when he was kicked in the head by a horse. One month later seizures began, which have lasted thirty-one years, gradually lessening in severity. Seizures come on in groups of short attacks lasting one to five days. Seizures are characterized by some fibrillary tremor, irregular clonic movements, tonic contractions with and without locomotor effect and violent rhythmical movements affecting the face, neck, shoulder and arm muscles and, to some extent, the legs. Muscles of the back are sometimes involved. The muscles involved are symmetrical but not synchronous in their clonic movements.

E. P. B., single, farmer, aged forty-two. Father healthy but of nervous temperament; no phthisis or inebriety, no nervous or mental disease in family. His mother was a healthy woman but during her pregnancy she had attacks of muscular twitchings and jerkings known as "shaking spells." These came on periodically during the day and even interfered with her sleep. They occurred in the latter part of the pregnancy during which she was carrying the patient. After the birth of the child the attacks disappeared. The patient had one healthy sister and lost one sister when she was a baby. He was perfectly healthy until his eleventh year. He was then kicked on the left side of the face by

a horse and knocked down, and remained unconscious for three-quarters of an hour. About one month later while sitting quietly in the house he had the first attack of his spasmodic trouble. These attacks would last with intermissions for twenty-four hours, beginning in the night sometimes and lasting till the next night. He would then go for a few days or a week without any, then they would come on again. At times he would have them with short intermissions of a day or two, all one summer, then they would go away for some weeks, then return again. His trouble continued in this way with various remissions till first seen by me, when he was thirty-four years old, at which time he had had the attacks for twenty-three years. During all this time he had no epileptic attacks and nothing resembling epilepsy. He had managed to get his education and attend to his work as a dairyman, spending some time occasionally at sanitariums for treatment.

The year before I saw him he had had an attack of sciatica affecting both legs. This lasted for a month. He had a second attack about four months before I saw him which had lasted all summer. On account of the pains in the legs and the other attacks, he had lost weight, being reduced from 158 to 105 pounds.

When first seen by me at my office he was not having the attacks, and presented the appearance of a nervous, rather anemic and badly nourished man. There was no objective trouble with the muscular system, though the muscles were rather poorly developed. There was some tremor of the hands, rather more in the right, and a little tremor in the legs. The dynamometer showed 30 for right and left hands. There was some fibrillary twitching of the muscles of the back. The skin reflexes were exaggerated. The deep reflexes were normal. There was no disorder in the sensory sphere, the special senses were normal. He had no anesthesia nor ataxia. Bladder and sexual organs were normal.

When the attack came on he was seized with certain rhythmical movements of the head; the hands would shake rhythmically, also the feet, and the lids would quiver. The muscles of the trunk were not involved, but the muscles of the back of the neck would contract and harden, pulling the head back slightly in clonic twitches. When called to see him in one attack at night I found his head moving rapidly antero-posteriorly. Also his arms would be shaken to and fro, but not synchronously, one arm would move more than the other. The trunk and leg muscles did not at that time move very much, though his eyes would blink and his feet and legs would be twitched in irregular clonic movements. He had no pain. The spells would last half a minute, then recur every minute or two, and in this way they would go on for one to five days. They would keep him from sleeping, un-

less he took some narcotic, then he would go to sleep and the attack would cease. During an attack there was considerable palpitation of the heart, he perspired profusely after the attacks and they would leave him exhausted. Consciousness was never disturbed.

The muscular movements of these attacks, therefore, consisted of violent tremors, tonic contractions of the body muscles without motor effect, clonic contractions irregular in distribution and fibrillary contractions of the body of the muscle which could be observed in between the attacks as well as in the most violent, to precede them. The attacks would occur in short intermissions, every half minute or so, and would last several minutes, and this would go on for from one to five days. These groups of attacks would thus recur every few days, sometimes every week or two, and sometimes intermit for several months.

The patient was not improved by the various measures employed by me, including hyoscine, hypnotics and tonics. He was hypnotized five times, without any special benefit. He was under my observation for one month. He then disappeared, and I heard nothing from him till a short time ago, five years after my first observation, when he wrote me that his general health was excellent and the nervous trouble very much milder, so that when the attacks came on he practically suffered little. He states that when they come on he goes to bed, as he is more comfortable there, and passes the time reading until they are over. He had had no epilepsy or hysteria. He is satisfied that it is a family disorder and accepts his affliction philosophically.

Case 3.—*Myoclonia of convulsive tic type*. Summary.—Male, aged forty, no heredity. Abscess of thyroid following typhoid fever; soon after developed clonic, tonic and fibrillary spasms, mainly clonic, affecting first and mostly muscles of abdomen, back and neck; less, those of face, and of forearm; least, those of lower extremities; cease during sleep; no pain; spasms are mostly coördinate, causing grimaces, wry-neck, movements of shoulders and trunk, isolated muscle spasm without locomotor effect, fibrillary and fascicular spasm not marked. Is able to walk and use his hands in delicate work. Spasms vary in intensity without absolute cessation. In four years has gradually improved. No organic disease, general health fair; no epilepsy, mental condition good.

R. M., aged forty years, traveling salesman¹. The family

¹Notes are by Dr. Joseph Fraenkel and myself.

history is negative and there is in particular no evidence of disease of the nervous or muscular system in the ancestry. Except for the usual diseases of childhood the patient has been well all his life. He denies venereal infections and has been a man of moderate habits. In November, 1896, he had typhoid fever, which kept him in bed for six weeks. The disease ran a mild course without serious complications. During its course, however, a swelling over the thyroid region began to form, which became an abscess. Eight weeks afterwards this abscess broke spontaneously, discharging pus. At the same time the right lower extremity became "awkward and lame," and the patient began to stoop. Shortly afterward he began to have what he called "spasms in the stomach." He was treated with hyoscyamin for this. Slowly the spasmodic discharges began to spread, and by March, 1897, they were very general. In May, 1897, he was treated with thyroid extract, and he says that he was delirious and out of his mind for four weeks. In August, 1897, another tumor of the size of an egg began to form in the thyroid gland, in the location of the previous one. This tumefaction was removed by Dr. Lilienthal, on October 5, 1897. According to a report received from the Mt. Sinai Hospital this tumor was a thyroid cyst. After leaving the hospital the patient says he was well and free from spasms for a period of ten days. They then returned and became continuous, so that he was admitted in August, 1898, to the Montefiore Home, having been ill for over one and a half years. During this time, the spasms, which began in the arms and abdominal muscles, had become generalized. The muscles of the tongue, face, throat and neck had become involved. He would thrust out his tongue and grimace, as in tic, or would throw his head back or twist it to one side, as in wry-neck. He had no pain at any time. Immediately after getting up in the morning the spasms were comparatively slight, but they increased in intensity and reached the climax about four o'clock in the afternoon. Upon going to bed the patient was compelled to take a hypodermic injection of gr. 1-100 hyoscine, and then had a good night's rest. Still at no time of the day, except when he was asleep, did the spasms cease entirely. They were worse when he was excited, and were mildest when he was quietly occupied. He was able to shave himself and to do various kinds of dexterous work with his hands.

Status præsens.—Patient is of medium size, rather poorly nourished, and his skin shows various tattooed figures. He is otherwise free from scars, except a small one over the thyroid region, slightly adherent to the deeper tissues. The skin is of a dirty hue and not very elastic. The muscles are fairly well developed; there are no localized or individual hypertrophies ap-

parent; the muscles are, on the contrary, when not agitated by the spasms, rather flabby and hypotonic. The usual attitude of the patient is a bending forward one, amounting at times almost to a complete doubling up of the body. The head is bent forward to the left and the chin turned to the right. When walking the attitude is the same. He seems to be drawn to the left and shows a sort of left-sided latero-pulsion. It is very difficult for him to assume the erect posture on account of spasms of the abdominal muscles, and attempts increase the spasms.

Temperature $99\frac{1}{2}$ (by mouth); pulse between 130 and 150; pulse is soft, volume rather small; respiration varies from 12 to 18, the rhythm and vigor being constantly interfered with by the spasms.

The thoracic and abdominal viscera are normal.

When the patient is observed, while sitting quietly on the chair, there is a ceaseless and varying play of muscular spasms. These spasms both affect individual muscles and physiologically connected muscle groups, and are essentially clonic, running often into a tonus. The muscles of the trunk, neck and face are affected most. The contractions of the extremities are very much less than those of the rest of the body and the upper extremities show more violent and frequent spasms than the lower; in fact, the lower extremities are, but for the occasional clonic spasms in the quadriceps, almost entirely free; in the upper extremities the muscles nearest the shoulder joint are affected most. The muscles of deglutition and mastication are also affected. There is frequent grinding of the teeth, and swallowing is sometimes interfered with, morsels dropping out of the mouth and fluid passing through the nostrils. There are at times noisy and irregular respirations, due to spasms of the diaphragm. After a number of these severe spasms the patient is covered with perspiration and is considerably exhausted. The frequency of the spasms varies from 10 to 60 per minute.

In the psychical sphere it is noteworthy that the patient cries easily; otherwise no defects are apparent.

Cranial nerves, special senses, gross motor power and cutaneous sensibility are absolutely undisturbed.

Pupillary and skin reflexes are good; tendon reflexes of moderate intensity and easily demonstrative when free from spasmodic contraction. Rectal and vesical sphincters are normal; the patient states, however, that he is unable to urinate except when he is sitting down, and that the spasms cease entirely during defecation and micturition.

The sexual functions are normal. The electrical excitability of muscles and nerves is rather increased, the direct stimulation

of the muscles is easily exhaustible. The mechanical irritability of muscles is slightly increased.

A *second examination* was made three months later. There is now a persistent tonic spasm of the abdominal muscles which holds the patient in a stooping position. The recti muscles are thus contracted nearly all the time. They relax only during sleep and for short periods during the day. The pectoral muscles show frequent, bilateral clonic twitchings, at times at the rate of about 50 a minute. Choreic, irregular, not bilateral twitchings are seen from time to time in the muscles of the arms; there is here and there a twitching of the biceps and triceps muscles alone, particularly on the left side. The platysma myoides shows clonic twitchings on the right or left side, but not simultaneously. The right sterno-cleido-mastoid muscle and upper trapezius undergo clonic twitchings and tonic spasm, pulling the head to the left, as in wry-neck. Similar spasms of the left sterno-cleido-mastoid muscle frequently occur, turning the head to the right.

On the whole, the left spinal accessory nerve is more affected than the right, and the spasms are more tonic on this side. Clonic tonic bilateral facial spasms producing prolonged (i.e., for two seconds) grimace occur, and along with them there occur irregular clonic contractions involving the tongue and face muscles.

The patient used to grind the teeth when asleep, but now there is a continual grinding of the teeth and smacking of the lips, as in forms of tic and chorea. There is no nystagmus or disturbance of ocular muscles.

The gluteal muscles go from a state of wavy, fibrillary twitching into a tonic spasm when the patient is put on his back (See Case I), while the irregular choreic contractions of the lower extremities occur at the same time. To mechanical irritation most of the muscles respond with myoid tremor.

Third examination, June, 1900, three and one-half years after the onset of the disease:

The patient's condition is somewhat improved. Still he is in almost constant movement. The spasms continue to affect chiefly the musculature of the neck, shoulders and upper arms, and trunk, the lower extremities being comparatively free; the deep muscles of the back are not affected as much as at the last examination. The number of convulsions per minute is variable, the finer fibrillary contractions occurring as high as forty; the coarser grosser movements occur as high as ten per minute.

The spasms are mostly clonic in character, but also fibrillary and tonic. These movements result in the flexion and extension of the forearms, abduction and elevation of the arms, a flexion of head on thorax, and very slight flexion of trunk on thighs. The

peripheral portions of all four extremities are least affected, the movements being greatest in the upper thoracic portions of the body and neck. The diaphragm is still irregularly affected, causing irregular breathing, and to some extent the speech, patient breathing and speaking in intervals. The movements are to some extent symmetrical as to muscles involved, yet not as to time of involvement. Contractions occur irregularly, affect the biceps, then jump to fingers, then to head, then to forearms. They affect usually coördinate groups of muscles, but also individual muscles. Fibrillary twitchings, involving very small limited portions of muscle, and twitching of distinct muscle bundles are seen, the rest of the muscle being apparently at rest. The movements of patient are well nigh continuous under observation, though when unobserved distinct intervals of quiet are apparent, though even then fibrillary contractions are evident.

Effect of position upon movements.—Recumbent posture diminishes movements of trunk, but to a less extent other movements.

Standing increases markedly movements of trunk and somewhat the movements of legs.

Noteworthy that muscles most concerned in standing are least affected, but upper extremities are threshed wildly about.

Standing on one foot possible for a length of time, equal to that possible to a normal person.

Standing with eyes closed, feet together is normal.

Fixation of attention steadies the spasm.

Volitional movements steady spasm, patient being able to shake himself and to do delicate manual work. Things do not drop from hands.

Irritation of skin.—Tickling, pinching, sticking, pressure and cold increase the twitching.

Emotion has a decided staying influence, he asserts.

Alcohol has no effect. Sleep inhibits the movements entirely. Spasms never rhythmical. Tonic spasms are apparent in anterior muscles of neck, yet upon closer investigation they consist of many rapidly occurring clonic contractions.

Sensibility.—No paresthesias. Pains.—In right side of neck and lower and upper extremities. Tactile sense, normal. Pupils equal and normal. Ocular movements normal, but affected by general spasms. Facial innervation equal. Pharyngeal reflexes diminished. No reflexes in upper extremities. Musculature fair in volume, no atrophies. Myotatic irritability increased. Myoidism excessive.

Abdominal reflexes lively. Cremasteric present. Knee-jerks markedly diminished. Achilles ditto. Plantar reflex present;

flexor response. Gross motility normal. No ataxia. Electrical examination: normal reaction.

Pulse 108, full. Respiration variable, 22. Marked prominence of superficial veins of upper extremities and abdomen (caput medusæ). Lungs normal. Heart: no murmurs. Liver apparently enlarged.

Fourth examination on March 25, 1902. The patient walks well, but his position is that of extreme kyphosis. There is continual nodding spasm of the head with occasional grimaces of the face. There are no spasms of the eye muscles. The neck muscles are occasionally contracted in tonic spasms which relax at the end of three or four seconds. There are continual rhythmic tonic spasms of the pectoral muscles, drawing the shoulders together, and of the abdominal muscles, pulling the trunk forward. The upper arms are affected in the same way, with occasional tonic spasms involving the whole musculature of the arm, so that there is no actual movement of the forearm. The forearms and hands are not affected by any spasms. There are movements of the back muscles drawing the scapulæ together. He has occasional movements of the intercostals and diaphragm, and owing to this he has, at times, difficulty in breathing. His spasmodic condition, has, on the whole, improved, but he has lost flesh and looks anemic and weak.

At the present time, 1903, seven years since the malady developed, he is much improved, but has still facial grimaces, and neck spasms like those of wry-neck. Mental condition good.

Case 4.—Myoclonia of type of degenerative chorea, or myoclonus-epilepsy. Summary.—In 1890, I reported in *Brain*, p. 71, a case of "Chronic Chorea with Autopsy." The patient, a male, aged eighteen years, negative family history, began to suffer with attacks of the chorea of Sydenham at the age of six years. These became chronic and associated later with epileptic attacks. For more than six years the patient suffered from attacks of chorea of a mild type, and occasional nocturnal epilepsy, which did not prevent him from going to school and afterwards to work. The movements increased in violence and extent, and he was finally, at eighteen years, obliged to give up work and go to a hospital. At that time he had not only clonic and choreic movements, but tonic contractions of the muscles of the trunk, fibrillary and wave contractions of the muscles, tremor and not infrequently attacks of an epileptic character. He developed pneumonia and died. I reported the case as one of chronic chorea with epilepsy, but as it appeared in the later stages it would certainly be classed

amongst the cases of myoclonus epilepsy, if that classification is adopted.

Case 5.—*Myoclonia of type of degenerative chorea. Summary.*—In 1894, under the title of "The Microbic Origin of Chorea, Case with Autopsy," *American Journal of the Medical Sciences*, January, 1894, I reported a case of a very singular character in a man aged thirty-four years, who had a good family history, who developed rheumatism in the tenth year followed by Sydenham's chorea. This chorea recurred every two or three years, until finally, eight months before he was seen by me, he developed violent choreic and clonic movements, affecting all the muscles of the body, with tonic spasms of the neck and back at times, and rhythmical movements of the head and arms. The case in these last stages resembled Case 2. He subsequently died and an autopsy was made. This man did not have any epileptic attacks, but had a combination of choreic convulsive tic, tonic and rhythmical spasms. The multiple myoclonus, however, was in the last year of his life the dominant feature of the case.

Cases 6, 7, 8.—*Cases of myoclonia of the type of convulsive tic.* In Thompson's "American System of Practical Medicine," under the head of "Choreic Tic" I reported a series of three cases of children who began with what appeared to be ordinary choreic symptoms. In these cases there was first a condition of ordinary choreic movements affecting the face and arms, later tonic and choreic spasmodic movements of the muscles of the neck, causing the patient to appear to have a combination of convulsive tic, or wry-neck, and chorea.

These three cases I called forms of choreiform tic, because beginning with a chorea, like that of the infectious or Sydenham's type, they later developed symptoms of wry-neck and had other tic-like spasms. They belong with the myoclonias of the convulsive tic type, like Case 3.

I have tried to determine the features of myoclonic disorders which ought to be brought out in a description and utilized in classification. Perhaps I have not all, but they seem to be about as follows:

1. The cause.
2. The association with other diseases.
3. The mode of onset.

4. The duration.
5. The persistence or remittance, or intermittance.
6. The groups of muscles or the limbs affected.
7. The symmetry, or bilateral synchronicity.
8. The quickness, or electric character of the spasm.
9. The involvement of muscular groups used coördinately.
10. The involvement of incoördinated groups.
11. The involvement of single muscles not under voluntary control.
12. The motor, or locomotor effect of the twitch.
13. The fibrillary twitching and the twitching of bundles or large masses of the muscle.
14. The predominance of 10, 11, 12 and 13, if several types exist together.
15. The association with tonic or rhythmical spasms, or with epilepsy and mental deterioration.
16. The association with pain, atrophy or paralysis.
17. The condition in sleep, or under anesthesia.
18. The control of the spasm by the will.
19. The influence of emotional excitement on the spasm.
20. The termination.

It is assumed that there is no organic disease and the trouble is not symptomatic of such condition.

Now applying all the tests of character to known and common spasmodic disorders, we find an agreement in the peculiarities of chorea, as it ordinarily develops, i.e., the infectious or Sydenham's type. Nor is there difficulty with symptomatic or degenerative chorea of Huntington.

I think that French and American neurologists also agree pretty well as to what is meant by the different types of spasmodic tic, tic *coördine*, or the tic neurosis of Collins. It includes, to my mind, not only facial tic, but torticollis, and many local tics, as of the diaphragm and larynx, of the trunk muscles, and it may even be generalized as in what I have called progressive or "choreic tic" (myoclonia of the tic type). It is degenerative in origin, and it is a cortical disorder. (I admit cornual or nuclear tics.) It is often a kind of occupation or habit neurosis. It is slow in onset, chronic in character, very nearly continuous in its manifestations. It usually affects the face, neck, the larynx, articulatory muscles, diaphragm or larynx, but it may affect the

legs. (I have reported such a case in a boy who had a jumping tic, as well as a speech and facial one.) It is not symmetrical in its distribution. The movements are lightning-like and cause locomotor effects.

There are with it usually no fibrillary twitchings or bundle movements. The clonus may, however, in severe cases, be prolonged into a tonus, and this is the tendency in severe cases. Rhythmical tremor and epilepsy may be associated with it. There is no pain, atrophy or paralysis, but rather a tendency to muscular hypertrophy. It ceases during sleep, it can be controlled in a measure by the will, and is made worse by excitement. It is only rarely curable.

The majority of reported cases of paramyoclonus multiplex can be placed in this group as myoclonias of the tic type.

There is another group of myoclonic disorders which has passed under the name of paramyoclonus multiplex.

Its characteristics, if there is such a disease, are claimed to be these:

It is caused oftenest by fright or some psychical disturbance.

Its onset is rather sudden and the duration rather chronic, i.e., months or several years. The spasms intermit, or remit. The muscle groups affected are mainly those of the shoulders, upper arms, thighs and trunk, and the face and periphery are rarely involved. Both sides of the body are affected, but not synchronously.

The movements are lightning-like, and the muscles affected are those of physiological groups, but single muscles and sometimes those not under control of the will may be affected. There is usually decided locomotor effect. There may be tonic and rhythmical spasm (Cases 4 and 5 or group I of Unverricht). The movements cease during sleep and are to a certain extent controlled by the will and increased by emotional excitement.

This type of disorder is distinguished from the tic neuroses by the intermittent duration of the spasms, the involvement of large groups of muscles producing bodily movements and tremors, the absence of spasm of small muscle-groups, causing unilateral facial grimaces and wry-neck movements. The rather favorable course. The neurosis should be placed among the

hysterical, or functional groups. My case (Case 2) shows it *may* be a family type and not associated with hysteria.

A fourth group of myoclonias includes myokymia and allied spasms. It is characterized by persistent fibrillary and wave-like contractions of the body of the muscles. Higher and more intense types of this condition show also clonus and tonus of individual muscles. The so-called Friedreich's myoclonus multiplex, the fibrillary myoclonus of Kny would go in this group. They are essentially intrinsic spasms, grosser kinds of fibrillary tremor. So far as I can make out Friedreich's original case was one of this kind, and I agree with Dr. Hunt that they are peripheral or spinal in origin.

Thus I would group the myoclonias under the head of:

1. Myoclonia of Friedreich, or peripheral type, including myokymia.

2. Myoclonia of the functional or hysterical type.

3. Myoclonia of the convulsive tic type, this last type being characterized by many associated spasms, choreic and tonic.

4. Myoclonia of the degenerative chorea or "familial" or myoclonus-epilepsy type, which is closely related with the third.

5. Myoclonia, of the type of the infectious and symptomatic choreas. Although there is sometimes a mixture or fusing of the above groups, they are essentially different disorders, and as a rule can all be easily recognized and easily distinguished from each other. All cases of paramyoclonus multiplex, reported as such, can be grouped in one or the other. Hence there is no need of that much misused term.

The Myoclonias and their Synonyms.—The following somewhat interesting list of myoclonias and choreas shows how extensive the nomenclature of muscular spasm has become. I have tried to assign them to the five groups above enumerated. Probably the arrangement is not perfectly correct, or complete, but it is approximately so. I have excluded rhythmical spasms because myoclonia means essentially an irregular spasmodic disorder, a muscle "tumult" (*klonos*), or twitch, or succession of twitches, not an orderly or rhythmical phenomenon like a tremor, though rhythmical spasm may be part of the general picture.

- I. Paramyoclonus multiplex, of Friedreich; astasic myoclonia,

of Vanlair; multiple spinal myoclonus, of Lowenfeld; fibrillary chorea, of Morvan; fibrillary myoclonus, of Kny.

II. Functional, or hysterical myoclonus multiplex, chorea major, chorea electrica, of Henoch.

III. Myospasmia; memory-spasms, of Friedreich; habit chorea; chorea variable des dégénérés; convulsive, or spasmodic tic; tic general; Tourette's disease, myriadrit; palmus; tic neurosis, of Collins.

IV. Degenerative chorea, hereditary chorea, Huntington's chorea; myoclonus-epilepsy; myoclonus, of Unverricht, of "familial" type; myoclonia congenita of Seeligmüller (?); hereditary degenerative chorea of Sachs.

V. Infectious chorea, chorea minor, Sydenham's chorea; chorea electrica of Dubini (?); chorea electrica of Bergeron; senile chorea of Gowers (?).

The stasic myoclonia of Vanlair is apparently a myoid tumor.

As there has been so much discussion over the nature of Friedreich's original case, I append here a translation of the history without adding the author's comments.

APPENDIX.

TRANSLATION OF THE ORIGINAL REPORT OF A CASE OF PARAMYOCLONUS MULTIPLEX. BY PROF. FRIEDREICH.

Ludwig Berlein, fifty years old, carpenter from Mosback (Baden) was taken ill in 1877, with sudden fever, and the appearance of a right-sided pneumonia, following which were developed a chronic induration with shrinking of the upper lobe of the lung, and a dilation of the bronchi of the same lobe. From that time on the patient suffered from dyspnea, chronic cough and mucopurulent expectoration. Lately, also, there appeared progressive emaciation and weakness, which interfered with his work, so that the patient was admitted to the clinic on January 17, 1878. Under the use of liver oil, morphine in the morning, strengthening diet and careful nursing the patient's strength and nourishment improved. Cough and expectoration diminished to a slight remainder, and the patient was able to return to his previous occupation on the 8th of April. But in the fall of the very same year the above-named phenomena returned with renewed violence, so that after a temporary stay in a few other hospitals the patient was for a second time admitted to the clinic on April 29, 1880. The symptoms of bronchitis in the region of the cirrhotic parts of the lung were developed to a high grade, while the lung itself did not expand any.

The left lung was perfectly sound. No fever. No change in the heart or abdominal organs. Two of the sick man's sisters were said to have succumbed to pulmonary tuberculosis.

During the first as well as during the second stay of the patient in the

clinic a most peculiar affection of a set of muscles in the upper and lower extremities became noticeable. This showed itself in the form of short rapid contractions, reappearing at short intervals, which affected the biceps and triceps of both arms, and supinator longus of the forearms exclusively.

At the same time chronic spasms showed themselves in the upper part of the thigh involving the vastus externus and internus and especially the rectus femoris, also to a less extent the abductors, and further the biceps and semitendinosus were affected, while the rest of the muscles of the upper part of the thigh were entirely intact. Here and there a muscle of the back or face was slightly affected by this disturbance, which confined itself to that particular muscle exclusively.

Here it was not merely fibrillary or fascicular twitchings but also spasms which involved the whole of the muscle which swelled forward in a mass, and bulged every time, even when the contractions of the muscles were not marked enough to produce a noticeable locomotor effect on the limbs to be moved. Only at times, at an unusually marked contraction, one could notice a slight change in the part to be moved, e. g., during many contractions of the biceps a slight flexion of the forearm or during some of the spasms of the supinator longus a slight supination of the hand.

The spasms of the rectus femoris produced a short and rapid forward movement of the knee joint, that of the biceps and semitendinosus a rapid movement of the part affected, following the tendons which limit the popliteal space.

The spasms, however, were unrhythmical throughout and varying in extent, even though they were separated from each other only by short intervals. (It may be mentioned here, by the way, that by placing the stethoscope on the affected muscles in the moment of their contraction a loud and clear sound, resembling the first heart sound in every respect, could be appreciated.)

Although the affection offered a perfect symmetry in its art, as the very same muscles were affected on both sides, yet every muscle showed a complete independence in relation to its individual contractions. Now it was this muscle, now that muscle, which twitched all for itself without any relation to the others, and if it sometimes happened that the contraction of a muscle on one side took place synchronously with another muscle of the same or other side, or even at times if two symmetrical muscles contracted at the same time, it was only an accidental coincidence, as it may be understood that such an occurrence was made possible by the frequency with which the spasmodic contractions appeared in each single muscle.

The frequency and intensity with which the contractions of each single muscle ensued were not the same on different days or at different times of the day, and in this regard a considerable difference could be noticed. During the times of greater agitation there were 40 to 50 contractions per minute in one and the same muscle, while during the hours of greater rest only 10 to 20 contractions could be counted. Only rarely did it happen that the spasm ceased entirely, or almost entirely, within one-quarter or one-half hour.

Were the spasms of less frequency, so were they also of less intensity, and vice versa. Frequency and intensity always stood in equal relation. It often happened that in one or the other muscle, contractions followed each other very quickly with increasing rapidity, and passed over into a short tetanus lasting about one or two seconds, which was always accompanied by painful sensations in the muscles. In general, the spasms were strongest and most frequent when the patient lay quietly in bed without carrying out any active motions of the extremities. Especially during the evening when the patient attempted to give himself over to the quiet rest of slumber, these cramps in the contracting muscles increased until they

produced painful sensations, and thus going to sleep was rendered difficult.

However, if sleep once set in, all the spasms ceased entirely while it lasted. Yet it often happened that the patient was frightened out of his sleep by a sudden very painful, crampy movement of both legs, so that the latter were jerked up against the abdomen. The adjoining individual contractions of the above-named muscles were then always of singular intensity, and it lasted hours until these quieted themselves. But many times, even during the day, although much more rarely than during the night, when the patient lay quietly in bed, there appeared flexion cramps of this sort in the lower extremities, similar to those that are known to appear in cases of acute myelitis.

As soon as one undertook real motions and prolonged active contractions of the affected muscles the spasms immediately disappeared, and the former movements were in no way injured by them.

Thus the patient was not at all disturbed at his work; as soon as the muscles weakened and began to rest, the spasms returned. So during the active extension or flexion of the forearm there were no traces of any spasms in the biceps and triceps of the same side, while the spasm of these muscles of the other quietly lying down continued without weakening.

The lower extremities were in analogous condition, only that in the thigh the painful contractions of the affected muscles continued also during upright position, but here also they entirely disappeared during locomotor actions, as walking. The rough motor power of the affected muscle was in no way affected, and only with the greatest effort could one flex an extended or extend a flexed extremity of the patient against the patient's will.

Station and locomotor coördination were very little affected.

The electric irritability of the affected muscles, as well as those innervated by the same nerve trunk, according to repeated examinations by Prof. Schultze with both kinds of currents, was normal throughout; the mechanical irritability also showed no deviation, and a stroke on the biceps, etc., either by a percussor or with the edge of the hand, showed an idiopathic swelling in no way higher or more prolonged than one often sees in most healthy individuals. On the other hand, there was an unmistakable increase of reflex irritability of the muscle to skin irritation. Even the effect of cool air on the skin of the exposed extremity increased the frequency and intensity of the spasm. On one occasion during a prolonged exposure, while the patient was being examined, the whole right arm was in painful and violent convulsive tremor, which lasted for one minute. The other skin irritations, such as pinching, pricking, etc., also increased the spasms, and as one would grasp the arm with his hand and squeeze firmly for some time, the spasms of the biceps and triceps would immediately become stronger and more frequent, and often followed each other in such rapid succession that they passed over into a short, lasting tetanus. Here, as it was mentioned above, there could be found no ground for the existence of an increase in the direct mechanical irritability of the muscles, and so this phenomenon was evidently the expression of an increased reflex irritability of the muscles. From the plantar surface one could also bring forth unusually strong reflexes of the lower extremities, and even a slight tickling was sufficient to call forth convulsive tremors of the legs, which repeated themselves after coming to rest a number of times. Tickling of the inner side of the thigh showed marked, although not unusually strong, cremasteric reflexes.

As far as the tendon reflexes were concerned, striking over the tendo-Achillis call forth only very weak contractions of the muscles of the thigh,

and no foot phenomena could be brought out, so that in this respect the normal relation existed.

In strong contrast to this there was a marked increase of the patellar reflex; even a slight tap on the tendon of the quadriceps-extensor or ligamentum patellæ was sufficient to produce the most lively and many times repeating extensions of the legs; and when one stretched the tendon of the quadriceps by pulling the patella down, the spasm of the affected muscle appeared, and there ensued painful cramps, which of themselves passed over into an intense tonus, during which time the extremity was strongly extended and the patella drawn forcibly upwards.

On the other hand, irritation and extension of the ligamentum patellæ by pressing it from below upward over the knee joint had no effect, nor were the spasms of quadriceps influenced by it in any way.

All these phenomena which have been described, as well as the muscular spasms and increased skin and patella reflex, were more marked on the right than on the left side. There were no symptoms which would refer to any vasomotor or secretory disturbances. The skin sensations remained normal in every way, and in the same way touch and pain, and differences in temperature were appreciated and localized quickly and precisely. Few phenomena appeared which could be interpreted to mean an injury to the deep muscular sense. There was no atrophy of the affected muscles, excepting the general emaciation and bad nourishment of the whole body, referable to the chronic lung disease.

The physical functions were normal; headache, vertigo, etc., bothered him most. The activity of the higher mental senses was also undisturbed. The pupils were of equal size, somewhat narrow, their reaction to light and accommodation active and normal throughout. There was no fever at any time. Pulse 80 to 90. Some complaints of palpitation. The digestive functions were undisturbed. The secretion and excretion of urine were normal.

The patient attributed the cause of these muscular spasms to a violent fright which he had five years previous at the bursting of a circular saw, and which affected him so thoroughly that he became "purely white" in his face, and at the same time he began to appreciate a sensation of weakness in his whole body; not until fifteen minutes had elapsed was he able to recover himself to any degree. At that accident, however, he received no injury or mechanical shaking up of his body.

About 14 days afterwards the patient noticed these spasms for the first time, and they grew worse later on, but they did not disturb him at his work. Besides that the patient said that he felt the combined sensations of pain and pressure in the arms and legs a number of years before that fright, always, however, only during rest, never during his work or any other active movements.

There was no hereditary predisposition to neurosis.

The patient during his double stay at the clinic was treated with liver oil, with special consideration of his lung disease and in behalf of his improvement of nutrition through which a favorable effect was intended, although the muscular affection remained the same. During the month of March, after only a few galvanizations undertaken by Prof. Schultze, the spasms rapidly began to diminish in intensity, and after a number of days disappeared without leaving a trace.

The increase in the skin and patella reflex diminished in quick succession, so that by the end of April the normal relations were restored in this respect also. The patient was discharged from the clinic by the middle of May.

CHRONIC PROGRESSIVE HEMIPLEGIA, WITH REMARKS ON
TWO CASES OF UNILATERAL PARALYSIS AGITANS
WITHOUT TREMOR.¹

BY HUGH T. PATRICK, M.D., CHICAGO.

M. K., eighteen years old, of German parentage, was first seen September 20, 1903. The family history is negative, both parents are living and well. She is the last of five children, there having been one miscarriage eleven years after her birth. The first three children died of diphtheria. The fourth child, born two years after the third, is living, three years older than my patient, and is strong but nervous. The patient was born at full term after a normal labor, walked at eleven months and talked at eighteen months, but not plainly until five years old. Excepting the ordinary diseases of childhood, from which she made perfect recoveries, she was well, strong and active until the age of thirteen or fourteen. Both the patient and her mother are emphatic in their statement that she had perfect use of all extremities until the beginning of the present trouble. She has never had convulsions, has developed mentally in a normal way, was bright as a school child, a rapid writer, and acquired unusual skill at fancy work. Menstruation began at eleven years, has always been regular and also painless until about a year and a half ago, when she had been working very hard. Since then there has been some but not extreme dysmenorrhea. At about thirteen she began to be rather irritable, impatient and emotional, as is the elder sister, and the mother attributes the change to the influence of this sister.

Shortly before the age of fourteen years a slight clumsiness of the right foot was noticed and writing began to cause fatigue of the forearm. After a few months the limp had become very apparent, the child was no longer able to run well and disability of the hand was distinct. At the age of fourteen years and three months, six to nine months after the onset, the patient left school, and she remembers that at that time she could still write well and do fancy work well, though with considerable effort. Gradually the weakness on the right side increased, but when fifteen years old she was still well enough to take a place as domestic with some acquaintances. She states that at this time she could carry a child on her right arm, and could use the right hand pretty well in washing and drying dishes, but was quite unequal to the task of buttoning her clothes or those of the children. Steadily the dis-

¹Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

ability grew and although her employers were unusually considerate, she was obliged to give up the place and go home in the spring of 1902, when seventeen and a half years old. Since that time her condition seems to have changed very little except as regards some symptoms apparently functional. As before noted, dysmenorrhea appeared just before she came home, and at the same time she became more irritable and emotional.

Soon afterward she began to notice that often on rising the neck was swollen, but would return to normal in an hour or two. This was not a mere sensation, as the mother says it was plainly visible. Indeed, this transient swelling, apparently of the thyroid gland, continues, and for the last three or four months the right side has remained slightly enlarged. At times the face flushes smartly, and at such times she has headache. These flushes are sometimes, not always, coincident with menstruation.

During the whole course of the trouble there has been no pain whatever, no tremor, jerking or convulsions, no dizziness, tinnitus or vomiting, no affection of vision, speech or mentality, no involvement of the sphincters, and the general health has remained good.

She is at present a ruddy, well-grown, well-developed woman, five feet five inches tall, and weighs 140 pounds. Mentally she is normal. Results of examination of thoracic and abdominal viscera, blood and urine were negative. The special senses, pupillary reactions, fundi oculorum and cranial nerves reveal nothing abnormal, except paresis of the seventh and motor root of the fifth as hereinafter mentioned. Nothing in the history or examination indicates inherited or acquired syphilis.

There is an incomplete right hemiplegia, and the patient states that it seems to her that sensation is not so good on that side. She also says that sometimes the right hand and leg feel numb, especially at the menstrual time, but repeated careful examinations have shown sensation to be perfectly normal in all its qualities, including the localization of sensations, the sense of position and of motion and stereognosis.

Voluntary movements of the right facial muscles are slightly less perfect than of the left. With the face at rest the right eyebrow is a little lower than the left, and a trifle of facial asymmetry is noticed, the right side being slightly smaller than the left. On raising the brows to wrinkle the forehead horizontally the left eyebrow rises a little higher, and in showing the teeth the left corner of the mouth is retracted more than the right. The eyes are closed equally, and no excess of associated movement of the right side can be demonstrated.

The muscles supplied by the motor root of the fifth nerve show more difference. When the patient bites the teeth strongly

together, masseter and temporal on the right side can readily be felt to contract less powerfully than on the left. Biting a pencil, she makes noticeably smaller indentations with the right side. Indeed, the patient asserts that on this side she can bite my finger only a little harder than I am able to endure. The pterygoids are clearly involved. Movement of the lower jaw toward the right brings the space between the canine and bicuspid teeth to the middle line, while toward the left the space between the lateral incisor and canine reaches the same point. When an effort is made to protrude the inferior maxilla straight forward beyond the upper, it deviates to the right to the extent of about three-fourths the width of the upper incisor. In opening the mouth *ad maximum* the middle point of the jaw makes a curving deviation toward the right, curving back toward the middle line on closing, but even at rest the interincisor space, or median line of the lower jaw is slightly to the right.

The right half of the tongue seems to me slightly smaller than the left, but it is not wrinkled or flabby, lies straight in the mouth, and is protruded straight. Neck muscles show no noticeable difference, except those going to the shoulder. The right shoulder is shrugged somewhat less powerfully than the left, and all movements at the shoulder joint are weaker on the right side. The arm can be elevated to an angle of about forty-five degrees above the horizontal. Flexion and extension of the forearm on the arm show more difference, pronation and supination and movements at the wrist are weaker still, while the greatest weakness is evident in the flexors and extensors of the fingers and in the small hand muscles. Grasp of the left hand is unusually good for a woman; with the right hand the dynamometer can be held, but the index cannot be made to move. Power to pinch between the extended fingers is nil between the index and middle, scarcely perceptible between middle and ring, somewhat better between the little and ring fingers. There is some power in the thumb, so that objects can be held after a fashion between thumb and forefinger. For instance, she can still manage to do some crochet work by holding the needle in the right hand and moving the work in the left.

In walking, the right arm is not carried flexed and pronated, as is so frequent in ordinary cerebral hemiplegia. It hangs by the side, and while it doesn't "dangle" vertically, as does the arm in advanced progressive muscular atrophy or poliomyelitis of the shoulder type, the hand does swing flabbily to and fro with every step, and gives one the general impression of flaccidity. The most pronounced feature of the gait is a decided limp on the right side. The gait is neither ataxic nor spastic, nor is there very marked "steppage," but foot-drop is plain and the outer border

drops more than the inner, so that in the forward swing the foot is in the equino-varus position. The patient toes distinctly neither out nor in. The right foot is raised higher than the left, it is not swung around in the arc of a circle, the toe does not catch the floor, but the heel does not come down first. Watching the patient walk one would say that the right leg was shorter, but careful measurement shows a scant one half inch difference. In the lower extremity the gradation of paresis is much as in the upper. That is, in a general way, the proximal muscles are strongest, those toward the distal end the weakest. Standing on the right leg the patient can flex the knee a little and straighten it again if she is steadied. Dorsal flexion of the foot is practically nil. With the patient recumbent the point of the foot can be moved upward not at all, but the toes can be very slightly extended. The calf muscles are good, but below normal. Standing on the right foot alone she can just manage, when steadied, to raise the heel from the floor. Inversion and eversion of the foot are impossible. Flexion of the toes is possible but feeble.

Aside from this deterioration of power toward the ends of the limbs, there is no localized paralysis; even no great preponderance in relation to physiological groups excepting as between the front and back of the leg. Flexors, extensors, rotators, adductors and abductors seem to be about equally involved.

The same applies to muscular volume. In no muscle or group of muscles is there unmistakable atrophy, but arm and leg on the right side are smaller in all their parts. The measurements in inches are: Arm, right, $10\frac{3}{4}$; left, 12; flexed, right, 12; left, $12\frac{3}{4}$; forearm, right, $9\frac{3}{4}$; left, $10\frac{3}{4}$; three inches above wrist, right, 7; left, 8; hand, right, $6\frac{3}{4}$; left, $7\frac{3}{4}$; over knuckles, right, $6\frac{3}{4}$; left, $7\frac{1}{2}$; calf, right, $13\frac{7}{8}$; left, $15\frac{3}{8}$; foot-arch, right $8\frac{3}{4}$; left, $9\frac{1}{8}$; foot-ball, right, $8\frac{1}{2}$; left 9; thigh, six inches above patella, right, $20\frac{3}{8}$; left, $22\frac{1}{8}$. Trapezius, deltoid and scapular muscles are also perceptibly smaller on the right. These differences are not such as are ordinarily found in anterior horn and peripheral disease, and, considering that the patient was less than fourteen when the trouble began, it seems reasonable to attribute them, at least in part, to lack of development. This hypothesis is supported by the discrepancy in the hands around the knuckles ($\frac{3}{4}$ inch) and in the feet ($\frac{3}{8}$ and $\frac{1}{2}$ inch), where there is very little muscular tissue. Skiagraphs also lend support to this view as they show the bones of the right hand to be smaller than those of the left. The electric examination indicates that lack of development would hardly explain all of the difference. While there is no reaction of degeneration nor even partial reaction of degeneration, there is certainly more change than is found in the cerebral palsies of children, lateral sclerosis or multiple sclerosis;

even more than I have ever been able to detect in cerebral hemiplegia with muscular wasting—so-called cerebral atrophy. The muscles showing the greatest diminution in size are the small hand muscles, the tibialis anticus and the peronei, and in these there is quite marked quantitative change.

Despite the general appearance of flaccidity the pyramidal tract evidently bears the brunt of the lesion. All the deep reflexes are greatly exaggerated on the right side, including the scapulo-humeral and McCarthy's reflexes. It is even easy to demonstrate that the right masseter jerk is greater than the left, and a short-lived supinator clonus is easily elicited. There is only an indication of ankle clonus, but Babinski's extensor response is typical, and may be obtained by pinching the leg as high as the middle of the calf. All reflexes on the left side are normal. Contractures are absent, with the exception of some slight shortening of the calf muscles. The foot may easily be brought to a right angle with the leg, but to get it much beyond this requires considerable force, and even strenuous pushing will not dorsally flex it to the extent that can easily be accomplished on the normal side. None of the excess of associated movement, so generally present in the cerebral palsies of childhood, can be found.

There is no trace of incoordination or tremor of any kind; no fibrillary twitching, no nystagmus, no speech defect, no dysphagia. Phonation is normal, and laryngoscopic examination shows normal movements of the vocal cords. As before stated, sensation is normal.

The apparent flaccidity, as shown by the dangling arm, foot-drop and moderate steppage, and the presence of real spasticity as shown by great exaggeration of the reflexes and Babinski's sign, present a very peculiar clinical picture. The case is entirely unique in my experience and an inconsiderable search of the literature has disclosed nothing just like it. Progressive muscular atrophy, amyotrophic lateral sclerosis, syringomyelia, multiple sclerosis, muscular dystrophy, neuritis, spinal tumor and hysteria may easily be excluded. It is difficult to imagine any gross cerebral lesion which could cause all the symptoms of this case and yet cause no sensory disturbance, no headache, vomiting, dizziness or optic neuritis, no more involvement of cranial nerves, no spasm, twitching, tremor or incoordination, no interference with speech and no mental deterioration. Degeneration of the upper motor neurones would seem best to explain the condition, but there is not nearly so much spasticity as in primary lateral sclerosis, and there is more diminution in muscular volume than I have seen in cases of that disease. I am unwilling to make a diagnosis. Perhaps I would better say I am unable, but I think the case more closely conforms to the clinical type proposed

by Dr. Charles K. Mills than to any form of disease with which I am acquainted, and his hypothesis of pathology, "a slowly increasing degeneration of the pyramidal fasciculi or of the cerebral motor neurone system," seems to suit the symptoms of my patient.

About three years ago Dr. Mills² reported a case which he called unilateral progressive ascending paralysis, and which he thought probably represented a new form of degenerative disease. As he stated in that report, the patient had been sent to him by me, and when I first saw the case just described, my first thought was that I had to do with a second example of the same affection. His patient I had not seen since July, 1900, eight months after his visit to Dr. Mills. To my written request for a further examination the patient quickly responded, and on reexamination I made a diagnosis quite different from that of Dr. Mills. My only surprise is that I had not thought of it before. I believe the man to be suffering from paralysis agitans, *unilateral* as to distribution (the so-called hemiplegic form), and *without* tremor. I believe this to be correct because all the *symptoms* except an attack of something like herpes can be explained by this diagnosis, and because I have now under observation *another* case almost exactly like it, in which the tremor is beginning to develop. Briefly, the case is as follows, my description agreeing essentially with that of Dr. Mills.

At the time of his first visit to me, September 26, 1899, M. J. P. was fifty-two years old, and the trouble had begun twenty months before. His wife first noticed that his right foot occasionally caught the floor or rugs, and before long he noticed it himself and made a conscious effort to lift that foot better in order not to stumble. After about eight months he was conscious of a dull ache in the extremity, and a few months later was sure that the right leg was not only more clumsy, but was weaker than the left—at any rate it felt weaker and was more easily fatigued. Five months before coming to me (fifteen months after the inception) he became aware that he carried the right arm against the side and crooked at the elbow, and soon afterward it began to grow a little stiff and was filled with a dull, heavy ache. Why this in itself did not suggest the diagnosis to me I am unable to comprehend, because only a year and a half before I had seen a gentleman sixty years old who complained of exactly the same thing (the leg being unaffected), which case I at once recognized as paralysis agitans, *sine agitatione*, a diagnosis confirmed by the subsequent appearance of all the classical signs. With the stiffness (in the case now under consideration) came some disability in the fingers. In writing the patient grasped

²Mills. *Journal of Nervous and Mental Disease*, April, 1900.

the pen too firmly, and in a few minutes hand and fingers would grow quite stiff and the arm would ache so that he could not continue. Buttoning clothing and other finger acts became difficult. Since that time the disability of the hand has been more prominent, at least more annoying than that of the leg. It has also been more progressive. Indeed, the patient affirms that the lower extremity is about as good now (April, 1903) as it was three years ago.

At the time of my first examination no record of measurements was made, but I noted the patient's statement that eight months previously the right thigh had been found to be one inch smaller and the calf one-half inch smaller than the left. Dr. Mills found a difference of one and one-fourth, and five-eighths respectively, and eight months later I found the differences to be one inch and five-eighths inch. The upper extremity showed no wasting. Aside from some increase in the disability of the hand, the present condition is so nearly that of three and a half years ago that the same description may serve for both, with such small exceptions as will be noted.

As regards the lower extremity, it is first to be noted that the disability is more apparent than real. To the patient there is a feeling of stiffness and clumsiness; to the observer the leg in action looks rather stiff and clumsy, but there is no spasticity, a scarcely perceptible paresis and no incoördination. On passive motion there is a little more resistance on the right side than on the left, but it is no more marked in quick or sudden movements than in slow ones—a nice distinction between the rigidity of paralysis agitans and that of pyramidal tract disease. The patient can stand on the left foot and easily pass the right over an object twenty-six inches high. He can run at fairly good speed, the only peculiarity being that he raises the right foot a little higher, and has not the same spring on that side; even hopping is well executed. He can stand on the right foot alone and let himself down until the thigh is at an angle of about forty-five degrees, and then rise so that he stands erect on the one leg, and he can stand on the right toes or on the heel. Especially the last I consider to be a severe test. Thigh measurements eight inches above the patella are eighteen and a half and nineteen and a half inches; calves, twelve and five-eighths and thirteen and one-fourth. In other words, the difference now is what it was more than four years ago.

Since the beginning, arm and hand movements have steadily but gradually progressed for the worse. The fingers are more clumsy, the arm and hand are weaker, the movements of even the larger joints less free. Putting the hand into the hip or even

side pocket of his trousers is a slow matter and requires some effort. To button the collar behind is almost impossible, and buttoning it in front is no easy matter. For six months he has been unable to button on the left cuff. The shoulder muscles are all strong, perhaps fractionally weaker than on the other side; the two biceps muscles seem to be equal, the right triceps not quite so strong as the left. Pronation and supination of the forearm are equal on the two sides, as are flexion and extension at the wrist. At the time of Dr. Mills' examination the grasp of the right hand was stronger than that of the left (180—160), now the right is weaker (85—95).^{*} Adduction and abduction of the fingers are slightly weaker on the right. As late as July, 1900, the right arm and forearm were slightly larger than the left; now the right arm is one-fourth inch smaller than the left; the forearms are equal. The same quick fatigue of the arm and aching on exertion remain. But the great discrepancy between the two hands is not in strength, but in freedom and promptness of movement—the disability characteristic of paralysis agitans. Apparently there is no movement of the fingers that cannot be executed, but no movement can be done quickly. For instance, movements of the fingers as in playing the piano can be executed with sufficient amplitude but only with extreme slowness. Smallest objects can be picked up with certainty but not quickly. This deliberateness of movement applies as well to muscles of the arm. Alternate flexion and extension at the wrist cannot be done rapidly, although as just noted, the strength is normal. The tooth brush is now used in the left hand, because with the right he cannot make the usual quickly alternating movements at the wrist. It is impossible for the patient to make a sudden movement, such as one would make in attempting to catch a fly. On command, a rectangular movement is very much slower on the right side. That is, if told to swiftly raise the hand from the knee ten or twelve inches, and then carry it ten or twelve inches horizontally one way or the other, the starting, the change of direction and the stopping take place slowly. This lack of prompt response to volition is less evident in movements at the shoulder. Last summer the patient played golf and could do so fairly well, but his swing was rather slow.

Movements of the face seem to be slightly better on the left side but the difference is so small as to be indeterminate.^{**} The frontalis acts equally on the two sides, but after some seconds of continuous action the right side tires a little, and the eyebrow drops a trifle. Masseters, temporals and pterygoids are equal.

^{*}My dynamometer is evidently much stiffer than that of Dr. Mills.

^{**}Since reading this paper I have examined a case of unilateral paralysis agitans in which the facial difference was much greater than in this one.

Achilles-jerk, knee-jerk, wrist-jerk and triceps-jerk are all brisker on the right side. An evanescent trace of ankle clonus can sometimes be elicited. Dr. Mills found patellar clonus. Apparently this varies as I have been unable to get it. The jaw-jerk is absent, as is Babinski's sign.

While comparing the muscular strength of the arms I was suddenly struck by a phenomenon that seemed to present the key of the situation. On demand the patient was forcibly supinating the left fore-arm against resistance, when I noticed that the

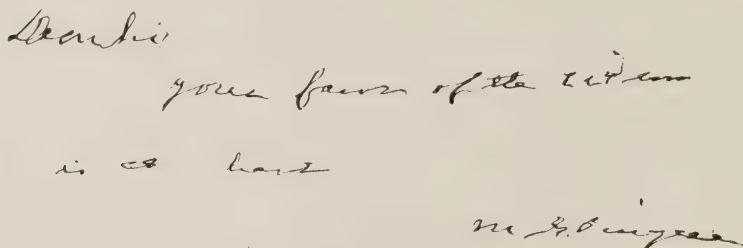
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<i>Tinct. colchici</i>	$3ss$
<i>Tinct. digitalis</i>	$3\frac{1}{4}$
<i>Morph. sulph.</i>	$grss$
<i>Potass. carb.</i>	$3\frac{1}{4}$
<i>Syr. toluatane</i>	$3v$
<i>M. S. Dose, one Teaspoonful</i>	
<i>Pingree.</i>	

Fig. 1. Written slowly and carefully.

right hand had assumed the position typical of paralysis agitans, and was shaken by a rhythmic tremor like that of this disease. The motion of this tremor seemed to be largely rotatory, that is, of pronation and supination. On retesting in the same way, the tremor was not so pronounced, but the motion was more a flexion and extension of the fingers, like that so often seen in paralysis agitans. I afterward noticed that the fingers are habitually carried in the position of paralysis agitans, though without tremor. For a long time, the patient does not know just how long, exertion of the right hand or arm has caused not only an aching pain but also tremor. This has been particularly evident during and

after some strain, as in buttoning the collar behind. At the present time this is very easily elicited by attempts to move the fingers rapidly as in playing the piano, and this tremor, though transient, is indistinguishable from that of Parkinson's disease. When the patient lies on the back and holds the right leg in a vertical position, it soon begins to ache, and is shaken with a rhythmic tremor affecting principally the quadriceps extensor. On the left side the same maneuver causes less tremor beginning later. When the patient forcibly raises the naso-labial folds and wrinkles the nose, there is an almost rhythmic tremor or jerking on the right side, the rate being about six per second. Tremor is quite evident in writing. If he takes plenty of time his penmanship is very good, but shows tremor in numerous letters. (Figure 1). If he tries to write rapidly, tremor is so bad as to make the product almost illegible (Figure 2). For several years



The image shows three lines of handwritten text in cursive. The first line is "Dear Sir", the second is "your favor of the 14th", and the third is "is as here". The handwriting is shaky and irregular, particularly in the second line where the words are difficult to decipher due to the tremor. The signature "H. T. Patrick" is written at the bottom right.

Fig. 2. Written rapidly.

a very annoying symptom has been a feeling of stiffness and drawing below the right occipital region, or in the right upper neck, leading the patient to frequently rub the place and forcibly move the head about in an effort to relieve it. Eighteen months ago the palm of the right hand began to perspire in excess. It is always moist, and last summer it often dripped sweat. Elsewhere there is no excessive perspiration. Lately, the patient has noticed that the skin on the back of his right hand is smoother, does not present the distinct reticulation seen on the other hand; looks more like parchment.

Sensation, coördination, stereognosis, special senses, fundi oculorum, mentality and speech are normal. There is no fibrillary twitching, intention tremor or nystagmus. Pain, headache, dizziness, vomiting, tinnitus and paresthesia have been absent. Sphincters are good and sexual power intact. During the last year a curious sequel of intercourse has been observed. Beginning ten or fifteen seconds after the termination of coitus, the right leg becomes rigid in extension, and is seized with a rapid

tremor or clonus lasting about fifteen seconds. This is unaccompanied by any pain like that of muscular cramp.

On January 18, 1903, the patient was put on 1-100 grain hyoscine hydrobromate *t.i.d.* Eight days later he reported that he was sleeping better, that the tense feeling in the neck was gone, and that the right side felt less clumsy. He could wrinkle the nose without the tremor formerly noticed, and forced supination of the left forearm, while causing the fingers of the right hand to assume the typical Parkinson position, induced no tremor. Writing was easier and caused less discomfort and tremor.

For purposes of comparison I wish to report in brief the following case:

T. K., a merchant, fifty-four years old, was referred to me December 30, 1901. Except the fact of rheumatism in the mother, the family history is negative. At intervals for twenty or thirty years the patient has had lumbago. Once it lasted two or three months. Twenty-five years ago he had rheumatism in the right ankle for some days. He has been somewhat troubled with hemorrhoids, is constipated, has taken a laxative every night for four years.

A year and a half before my examination he began to have pain about the middle of the right arm whenever he put the hand into his hip pocket, sometimes on other backward movements. This lasted for ten months, from May, 1900, to March, 1901, and during part of this time the middle joint of the right index finger was swollen and tender. During the following summer, 1901, the right upper extremity became weaker—as the patient expressed it. That is, in writing the hand soon became fatigued, and in rowing the right arm and shoulder tired before the left. Like the preceding patient, he began to carry the arm semi-flexed, and it felt rather rigid and clumsy. The disability gradually increased, writing became more difficult, and caused a tired ache in the arm and shoulder. A very short time before coming to me the right knee had begun to feel slightly rigid, and for a few days he had been annoyed by the heel occasionally catching the ground. In the upper part of the neck on the right side was a distressing sensation like a drawing or pressure.

Examination revealed a state of affairs very much like that of the preceding case: On the right side a plastic rigidity, unlike that of pyramidal tract disease, like that of paralysis agitans; slowness and clumsiness of movement, no real paralysis; the right extremities slightly smaller than the left, and the deep reflexes of the right leg more active than those of the left. There was no tremor except after some physical exercise, when the whole right arm trembled. The fingers of the right hand were habit-

ually carried in the position typical of paralysis agitans, and he presented the characteristic facies of this disease.

Since that examination his condition has changed very little, except that the leg is slightly more involved, the foot catches the ground more frequently, and he walks with a slight "hitch" on the right side. He left his business and has taken excellent care of himself. Beginning about December 1, 1902, he has noticed that the left elbow seems rather stiff and a little sore, especially when the arm has been for a long time in the same position. Strong efforts at pronation, supination, or flexion of the left arm against resistance cause an accentuation of the Parkinson attitude of the right hand and generally some tremor.

In this case there would seem to be no cause to doubt the correctness of the diagnosis, paralysis agitans. But excepting the facies, the other case, the one examined by Dr. Mills, presents almost exactly the same symptom-group: the same ache and fatigue on exercise, the same slowness and clumsiness of movement without paralysis and without ataxia, the same way of carrying the arm, the same position of the fingers, the same sort of discomfort in the suboccipital region on the affected side, the same sort of tremor induced in the same way, the increase of deep reflexes and diminution of muscular volume on the affected side. That one began in the leg and the other in the arm seems to me to be important. I agree with Dr. Mills that the attack of more severe pain and skin eruption (herpes?) of the first case does not constitute an essential feature. Unilateral distribution of paralysis agitans is the rule in the incipency. Marked unilateral paralysis agitans without tremor is unusual, but it is far from being unrecognized in the literature. A greater difficulty in these two cases, especially in the first one, M. J. P., is the combination of diminished muscular volume and increased reflexes. After all, the former symptom is very inconsiderable in degree, and while the latter is not the rule in paralysis agitans it is not rare. In my opinion, lack of use is too often proposed to explain reduction in size of an extremity, and I am not prepared at present to assert that this will account for the difference of one inch in the thighs and five-eighths of an inch in the calves, but I do think that it is quite as reasonable to thus explain it, or to simply say that it is an unusual manifestation of paralysis agitans as it is to invoke the aid of a new and not yet understood disease. Indeed, Jolly³ speaks of the diminution of muscular volume which ordinarily appears in the course of paralysis agitans. Besides, degeneration of the pyramidal tract alone would explain it no better. The appearance of tremor during or immediately after exertion, and in the involved but not trembling extremity on forced activity of

³Jolly: *Handbuch of Ebstein and Schwalbe*, Vol. IV, p. 865.

the other side, are manifestations of paralysis agitans often noticed in incipient cases or old ones without tremor.

Ten months after Dr. Mills' report Dr. Spiller⁴ published another case under the same title, but I am inclined to think that the disease is not the same. His case showed distinct spasticity, ankle-clonus and Babinski's sign, but was peculiar in presenting optic atrophy on the affected side. Furthermore, the patient occasionally found some difficulty in retaining the feces. Judging from his report I should say that he probably had to do with some affection different from any of the cases in this report.

To summarize, I would say that my first case apparently belongs to the clinical type indicated by Dr. Mills, but that my second case, the one reported by him, certainly affords no basis whatever for the creation of a new type, as it is an example of unilateral paralysis agitans without tremor.

⁴Spiller: "A Case of Unilateral, Progressive, Ascending Paralysis." Philadelphia Medical Journal, February 9, 1901.

THE WORK OF A NEUROLOGICAL DISPENSARY CLINIC.*

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During the year 1902 there were 1,780 new patients suffering from some well-defined form of nervous disease who presented themselves for treatment at the Vanderbilt Clinic. Of these 953 were females and 827 were males. This number does not, however, represent all of the new cases applying to the clinic for treatment. This number was 2,044, 1,068 being women and 976 men. At least 134 of these did not present symptoms referable to the nervous system, and in 130 patients no definite diagnosis could be made. The total number of visits made by these patients was 10,121, thus making an average of about five visits per patient. Inasmuch as many of the patients come once only it is probable that the average number of visits is between seven and eight. A number of patients came once a week throughout the year. In this connection it is of interest to note that a few patients suffering from slowly progressive affections have been coming to the clinic almost monthly ever since the clinic was opened in 1889.

Contrasting these figures with those of the first year of the clinic's opening, it is found that the number of patients who have come for treatment has been doubled. Thus in 1888-1889 there were 944 patients or, for the first twenty-three months of the clinic's opening, there were 1,888 cases, less by 200 than the number for the twelve months of 1902 alone.

Mental Diseases.—In attempting to present an analysis of this material we have deemed it of interest to consider at first the purely mental diseases that have come for diagnosis and treatment. The commonest forms of mental affection presenting themselves have naturally been in children. In these deficient development is represented by idiocy in six cases, imbecility in 54, making a total of 60 cases or an incidence of about three per cent.

*Report of Clinic of Dr. M. Allen Starr, 1902.

It may also be of interest to note here that the Board of Education of this city has already taken steps to establish supplementary schools for backward children (called secondary schools on the continent of Europe). A few schools for this class are in successful operation, being maintained at other hours in the same school building as the regular schools. Obviously some systematic examination should be made in every child dismissed from the regular school system by school teachers or medical inspectors because of its mental deficiency. Such examination should be made by properly qualified physicians appointed by the Board of Education, or, as Dr. Starr suggests, the city might be divided into districts and the schools refer their cases to the nearest neurological clinic. As to whatever arrangements are employed all agree upon the necessity of supplemental schools, and the importance of systems of medico-pedagogical training for the defective.

The insanities are not well represented in dispensary work. In men, general paresis was the commonest affection, 23 (one per cent) presented unquestioned evidence of this affection, and as would be expected, always in the early stages. There were no women with paresis, which seems rather peculiar, inasmuch as syphilis, the most important etiological factor of paresis, is almost equally common in women, particularly in the lower strata of society. Probably the less exacting symptomatology for general paresis in the past few years accounts for the increased number of cases for this year's statistics, and for the apparent increase of the disease in general during the past decade. At present the signs of cerebral syphilis, plus positive evidence of mental alienation, are considered by many as sufficient data for an early diagnosis of general paresis.

Taking into account the essentially systematized and persecutory nature of the fixed delusions in paranoia, and the personal well-being of such patients, it is surprising that as many as seven men and one woman presented themselves at a neurological clinic. In most instances the patients come to the clinic for the secondary development of distressing hallucinations of hearing. The occurrence of melancholia in six men and in 24 women should be considered in the light of the older classification of the disease and not in the restricted sense of Kraepelin as a type of insanity of

the involution period. In other words it includes manic-depression as well as true melancholia.

Senile dementia was diagnosticated in three cases. There was one patient recorded suffering from mild puerperal mania.

Cerebral gumma, and cerebral syphilis with mental symptoms, were diagnosticated in six instances. Therefore, the total number of patients with mental trouble was 131, or seven per cent. This is not a great number in a large neurological clinic when one recalls that patients suffering from mental disorders prefer to visit more or less voluntarily a clinic of this type, which, of course, does not possess detention wards or even hospital wards of any sort, a feature which should not be lost sight of in establishing out-clinics with psychopathic hospitals in close connection with large neurological dispensaries.

Nervous Diseases (Functional).—Dividing the neurological cases into those usually classed as functional and those organic, there were 873 (49 per cent) of the former, and 776 (44 per cent) of the latter, distributed as the following brief record indicates.

Neurasthenia is par excellence the disease of dispensary patients. A more complete analysis of these cases is contemplated in a further communication. Here it may be said that there were 416 (23 per cent) patients who presented themselves for treatment, in whom this affection was diagnosticated, 250 in men and 166 in women. Apart from the marked sexual element in 62 male cases and 38 menopause neuroses, the cases of neurasthenia presented no particularly interesting features. The foreign population contributed over eight-tenths of the neurasthenics. Among men the Russian Jews preponderated.

Hysteria is given as the diagnosis in 80 cases. There was a history of major hysteria in but three cases. The remainder represented badly trained individuals of unstable nervous organization.

The *convulsive disorders* were represented by 135 (19.5 per cent) cases of chorea, of which 128 were of the usual type of this disease. There were five cases of chronic chorea and two patients with hemichorea, i.e., cases of chorea of the latter class which remained persistently unilateral.

Epilepsy, next to neurasthenia, contributed the largest incidence with 180 cases (ten per cent). An analysis of these cases is contemplated elsewhere.

Convulsive tics were not common, there being only 24 cases, about one per cent, of which 15 were of the ordinary type of spasmodic tic. Seven were obviously of the early type, and six were of tic douloureux. Women preponderated in the ratio of two to one.

There were 15 stammerers. The proportion of stammering in the general population would from these figures seem to be very small, but in view of the fact that we have no definite means of determining the general proportion of stammerers, some figures as recently published by M. Chervin of Paris in a monograph may be of interest here. As stammering is a cause of exemption from military service, it is possible to determine its frequency in different continental nations. It appears that of 1,000 conscripts examined the number rejected is as follows:

	Per 1,000
France	7.50
Switzerland	3.23
England	2.87
Austria-Hungary	2.20
Belgium	2.10
Italy	0.86
Russia	0.19

Comparing these numbers with those that represent the frequency of nervous affections, he finds that in France the nervous affections are exactly twice as numerous as the cases of stammering; in Belgium they are four times more numerous, in Italy seven times, in Switzerland eight times, and in Russia thirteen times. No statistics of stammerers in the United States are given.

There were three patients with motor speech defects, and three patients with simple idiopathic tremor.

Paralysis agitans showed 17 cases, eight men and nine women. They did not present any features apart from the usual types.

The etiological statistics of the disease throw no new light on its pathogenesis.

Organic Lesions (Peripheral Nerves).—The peripheral nerve lesions of one type or another were numerous. They represented 379 cases (21 per cent), distributed equally between neuralgias 145 cases, neuritis 117 cases and peripheral palsies 117. Of this large number of peripheral nerve cases men were affected in 244 and women in 135 cases, showing the greater incidence of men to this type of lesion.

Of the neuralgias, sciatica was the most common, 48 cases; supraorbital in 16 (9 male and 7 female); intercostal 14 (3 male, 11 female); facial 25 (15 male, 10 female); generalized neuralgia in 18 (13 male, 5 female); brachial in one, coccygeal in one, ulnar one.

Neuritis was general in 12, traumatic in 18, lead in 11, alcohol in 7, occupation in 8, arthritic in 6, diphtheria in 3, typhoid in 1, circumflex 1, ulnar 1.

Peripheral palsies were represented by 117 cases. Facial palsies were most common in 41 (17 male, 24 female); musculo-spiral in 35 (29-6). Erb's palsy was represented by 5 cases, the ulnar nerve in 5, circumflex in 1, lead palsy 5, larynx 1.

Affections of the spino-peripheral neurone were present in but 46 cases. Of these anterior poliomyelitis was diagnosed in 24 cases. With reference to the incidence of these cases, 16 cases showed an initial history of fever, headache and vomiting. There was a history of initial convulsions in one case, and no recorded histories in seven. On the question of causation the history is negative in 16 cases, and given as due to a fall, measles, infections, dentition and pneumonia in 1 case each. The months of invasion are of interest. There was 1 case in January, none in February, 1 in March, none in April or May, 1 in June, 6 in July, 8 in August, 2 in September, none in October or November, 1 in December and no date given in 4.

Chronic anterior poliomyelitis was present in six cases, presenting the picture of progressive muscular atrophy. These were all in men.

Ophthalmoplegia was present in 11 cases. These patients were all men.

Tabes dorsalis was present in 32 patients. Only two of these were women.

Of these 32, record of 25 was kept—all males. The following occupations were represented: one each, driver, carpenter, farmer, soldier, bookkeeper, tailor, stone-cutter, fireman, janitor, butcher, lithographer, sleigh-maker, musician, cigar maker, butler; 2 bar-keepers, 3 salesmen, 2 cooks, 2 laborers. The earliest age at onset was 28, the latest 49, although one patient is put down as 14 years of age. It is doubtful if this early case is one of tabes. The average age of this series of 25 is about 35 years.

Bearing on the question of etiology, it was found that 14 had had syphilis, two had led extremely dissipated lives and nine presented no evidences nor history of syphilis, nor other etiological factor. In ten of the 14 presenting syphilis the initial lesion had been acquired 20, 8, 10, 28, 26, 18, 13, 30, 15, and 19 years previously, respectively. No special hereditary factors were present in any. In one there was senile dementia, in another grand mal epilepsy, in another tuberculosis, in the parents.

A few interesting facts concerning the earliest symptoms may be recorded. There were, as earliest symptoms, neuralgic pains in 11, perforating ulcer in 1, gastric crises in four, general weakness in the legs in 2, blindness in 4, ataxia in 3, and myosis in 1. The knee-jerks were absolutely lost in 24 and diminished in 1. The Argyll-Robertson pupil was present in 21 instances. There was normal light reaction in 3; myosis and optic atrophy in 1. Other detailed analyses are left for a special paper.

Friedreich's ataxia was found in 3 patients, 2 men and 1 woman.

Lateral sclerosis was present in 6 men and one woman.

Amyotrophic lateral sclerosis was present in 3 cases.

Multiple sclerosis was found in 6 men and in 4 women.

Myelitis was present in only two cases, certainly bearing out the recent contentions of Starr, Gowers and Risien Russell, that myelitis is really a rare affection.

Ataxia paraplegia was present in 5 men, and spastic paraplegia in 2 men.

Tumors of the spinal cord were present in two patients, one man and one woman.

Brain Affections.—Organic diseases of the brain were observed in 85 cases. Eighteen of these were in children presenting the symptoms of infantile cerebral palsies. Causation is here represented as whooping-cough in one, trauma in two, prolonged labor in four; the rest were negative. One patient was diplegic. Monoplegia was present in two men and two women. Hemiplegia was present in 58 patients, an incidence of 2.7 per cent. Forty-three in men and 15 in women. Two patients had aphasia. Tumor of the brain was present in four cases, three men and one woman; two cases of cerebral gumma were diagnosed.

Syphilis of the brain was diagnosed in 12 cases (3 men, 9 women).

Vascular Lesions.—Vasomotor affections were diagnosed in 16 patients, angioneurotic in 4, flushings in 3 and erythromelalgic symptoms in 9 patients, 6 men and 3 women.

Exophthalmic goiter was present in 8 women.

Miscellany.—Alcoholism in 43 males, 1 female; muscular dystrophy, 3 males, 1 female; atheroma 3; arteriosclerosis 12; concussion of brain 3; fracture of skull 1; headache, 10 males, 75 females; hemicrania 2; herpes 1; hemianopsia 1; insomnia 2 males, 11 females; pachymeningitis 4 males, 1 female; optic atrophy 2; paresthesiæ 5 males, 11 females; senility 1; incontinent bladder 1; local atrophy 1.

Society Proceedings.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

April 28, 1903.

The President, Dr. H. A. Hare, in the chair.

Poliomyelitis in the Adult Involving All Four Extremities.—Dr. John K. Mitchell exhibited this case. C. R. Wadley, aged twenty. There is nothing in the history, either personal or family, of interest. The boy was in ordinary health and has never suffered any injury until August 15, 1901, when he suddenly suffered for a day or two with stiff neck and pain in the back. Two days later he lost consciousness, according to the account which he has given, and remained unconscious for twenty-four hours. He had fever for a week following, but no vomiting, diarrhea, or convulsions.

On recovery from the fever he was completely paralyzed and could not move his head. Sensation remained perfect. Motion gradually returned, first in the head, then in the upper right arm, then in the lower left arm, then in the upper left leg and then in the lower right leg.

In the autumn of 1902 he was admitted to the Orthopedic Hospital. He was then entirely unable to stand. He could use his hands very little and could not raise his legs from the bed. When he endeavored to sit up, he was unable to get his spine straight from weakness in the back muscles. The worst parts at that time were the lower right arm and hand, the upper left arm, the upper right leg and the lower left leg. The affected parts were very cold and presented slight contractures. He had no knee-jerk and the plantar reflex was very small, cremasteric reflexes were absent and the abdominal slight. There was fair reaction to faradism in the arms; very poor in the legs. There was no degenerative reaction, KCL greater than AnCL everywhere.

He has improved very slowly, but steadily, and the only member which seems unsusceptible of betterment is the right hand, in which the destruction of the intrinsic muscles has been so complete that he has very little use of it, and very little prospect of a valuable member. In every other way there is a change for the better, and the boy is able to walk, supported in the wheeled-crutch, and can take a few steps with assistance even without the degree of support which this apparatus gives.

The case was shown as an interesting example of widespread and irregular distribution of a poliomyelitic paralysis at an age when patients are commonly exempt.

Dr. A. A. Eshner referred to a typical case of anterior poliomyelitis in a boy seven or eight years of age involving all four extremities. At the same time he had under observation a boy five or six years of age with flaccid paralysis of the extremities who was at first thought to be suffering from anterior poliomyelitis, but who later exhibited derangement of speech and incoördination of movement which led Dr. Eshner to believe that the trouble was of cerebral origin.

Dr. H. A. Hare said that there had been admitted to his ward in the Jefferson Hospital, six or seven weeks ago, a woman about nineteen years

of age, with the statement that she was convalescent from typhoid fever and was universally paralyzed. There was a history of five weeks' fever which careful questioning reduced to two weeks. The temperature had ranged as high as 105° F. When admitted she was absolutely paralyzed in the upper extremities and could slightly move one leg. There was no interference with speech or intellect. The hands presented a rhythmic tremor resembling that of chorea as it is seen in the dog. The emaciation was marked. There was exaggeration of the reflexes both at the elbow and at the knee, but not in the feet. Under careful nursing, massage and tonics the patient rapidly improved so that in ten days she could move all four extremities. At the end of that time she, unexpectedly, left the hospital and has been lost sight of.

Dr. John K. Mitchell believed that one reason why the limit of probable beginning improvement was within one or two years was because in bad cases the unaffected muscles atrophied from disuse. As showing that even very old cases are not insusceptible to improvement he referred to a case of fifteen years' standing in a girl eighteen years of age. When she came under observation there was almost complete paralysis of both legs. She was able with difficulty to shuffle across the floor a distance of thirty feet. She is now, after a year's treatment with massage and electricity, able to walk a mile with the aid of a stick.

A Case of Brachial Monoplegia of Spinal Origin.—This report was made by Drs. W. G. Spiller and T. H. Weisenberg. The patient had been in the service of Dr. Spiller at the Philadelphia Hospital. The clinical history was very incomplete, but there was sufficient to show that the man had had a paralysis of one upper limb. An area of sclerosis was found in the lower cervical region in the lateral column and anterior horn on the side corresponding to the paretic limb. This sclerosis was evidently the result of thickening of the blood vessels of the affected part. The case was important as an example of monoplegia of spinal origin in an adult.

A Case of Thomsen's Disease, Associated with Pseudo-muscular Hypertrophy.—This case was reported by Dr. Horace Carncross. The patient, a young man aged twenty-one years, was troubled with rigidity after resting. At birth he was a large child, weighing fifteen pounds. When nine months old he weighed forty-two pounds. When he was two years of age stiffness of the hands was noted and later of the legs and other parts of the body. If he fell he could not get up until after the lapse of five minutes. At this time he was very strong. At the age of six years he went to Girard College. From ten to fifteen years of age he was not as strong as before, or as his development would indicate. At the age of seventeen he began to waste. He has great difficulty in getting up in the morning. In going up stairs the first two or three steps give him much trouble, after that he goes up more easily, and at the second flight he can run. All the muscles are rigid, including those of the tongue.

The sensory symptoms are negative. The patellar reflex and biceps-reflex are absent, except on reinforcement. The electrical reactions are normal, except that the muscles respond sluggishly. The eye-grounds are normal, and the pupils react normally. The family history is negative, except that the mother had recurrent attacks of what were spoken of as facial paralysis, but which from the history were more probably attacks of facial spasm.

Dr. F. X. Dercum referred to a case which he had presented before the Society some years ago,—that of a bricklayer who when he started to work would suffer from a tonic spasm of the muscles. In that case there was no family history suggesting Thomsen's disease. He had merely termed it a case of myotonia. The present case he considered an anomalous one, but he thought that it was a muscular dystrophy associated with myotonia.

He believed that a study of these cases showed that our conceptions of the various types of dystrophy needed widening and readjustment.

Dr. D. J. McCarthy referred to three cases of Thomsen's disease which he had seen in Jolly's clinic in Berlin. In two of those cases the symptoms were no more marked than in the case shown tonight. The occurrence of facial spasm in the mother, and the fact that in the patient the condition had been present from birth, he considered of interest.

A Note on Periodic Insanities with the Report of Three Cases of Intermittent Melancholia.—Dr. Alfred Gordon read this paper.

Dr. F. X. Dercum said that mania, melancholia and circular insanity were essentially recurrent diseases. The practical point brought out by the paper was that irregular, intermittent melancholia was unfavorable as to prognosis. Ordinary melancholia yields a more favorable prognosis, but the attacks are liable to recur after rather prolonged intervals. Our suspicion should always be excited when the onset of melancholia is sudden and the recovery abrupt. It usually means recurrence after a relatively short interval as compared with ordinary melancholia.

Dr. John K. Mitchell referred to the fact that even in ordinary melancholia, recovery may occur suddenly. The line separating simple melancholia and the recurrent variety is very indistinct.

Senile Dementia.—This paper was read by Dr. Pickett.

Dr. F. X. Dercum regarded the present admirable communication as a worthy companion paper of Dr. Pickett's previous communication on dementia præcox. The paper was full of suggestions and brilliant generalizations. Some were quite startling, while others were in keeping with what we should expect.

Dr. Charles K. Mills said that with regard to the occurrence of other types of insanity than senile dementia in old age, he was quite sure that this was the case, and had seen a few striking illustrations. He referred to the case of a man eighty years of age who during his previous life had had at least five or six attacks of melancholia. He had retained his general mental vigor. At the age of eighty years he developed a typical attack of melancholia of the same type as he had previously had, although perhaps more severe.

Periscope.

CENTRALBLATT FÜR NERVENHEILKUNDE UND PSYCHIATRIE

(15 January, 1903.)

1. On the Limits of Psychiatric Knowledge. R. GAUPP.
2. On a Peculiar Katatonic Symptom-Picture Removed by an Operation.
K. BONHOEFFER.
3. Ophthalmoplegia Interna Traumatica. ERNST SCHULTZE.
4. The Trend of Investigation in "Psychologic Studies." W. WEYGANDT.

1. *On the Limits of Psychiatric Knowledge.*—Gaupp says: Our science is not merely a branch of natural science, but the symptoms to be studied belong mainly to another field of labor, not entered by the explorer of natural science. It is a mistake to believe we can localize the psychical elements and thus make the psychical processes understood. Pathological histology will never do much for general psychopathology, especially not regarding the laws of psychical phenomena of healthy and sick persons. There is no possibility of understanding psychical events by their material bases, which we do not know in individual cases. Chemical science too cannot explain the psychical phenomena,—it being nothing else than molecular mechanics, which cannot be compared with psychics. The facts of anatomy and chemistry not helping in the investigation of psycho-pathological phenomena, our etiological knowledge is defective, and a difference of opinion arises regarding the axiom that like causes have like effects. It is said, for instance: Because one drinker suffers from delirium tremens, a second one from mania with jealousy, a third one from Korsakoff's psychosis, it cannot be that the same causes produce the same effects. However, not one cause alone, but several of them result in psychoses; we may recognize only one, and so this one may appear to have several effects. These difficulties lead to the conception of exogenous and endogenous causes of psychoses; some authors speak about predisposing and exciting causes. We have made some progress regarding the exogenous, but less about the endogenous causes. The expressions "degeneration" and "psychopathic disposition" show the uncertainty and vagueness of our opinions.

We know very little as to the individual psychological attributes of the predisposed and their specific affinities for certain diseases. We do not know the happenings in the brain of the healthy, the predisposed and the diseased. Therefore we are often unable to appreciate what the real cause is, and what it only seems to be.

The laws of conservation of energy and psychophysical parallelisms are applicable within the psychical sphere. The question is: Can science discover that application? Experimental psychology can explain many psychical phenomena. Self-observation combined with the observation of others is the best method of investigating psychical phenomena. We have made little progress during the last decades in the scientific exploration of psychological disturbances. There has been a dearth of psychology, of the knowledge of psychical casuistics. Pathological anatomy has become the only hope of the future, according to the general belief; and this the writer endeavors to combat by his explanations.

2. *On a Peculiar Katatonic Symptom-Picture Removed by an Operation.*—Bonhoeffer declares that so-called katatonic symptoms are not pathognomonic of a special disease. Negativism, flexibilitas cerea, verbigeration,

rhythmical movements, and stereotpy are observed in diseases altogether unlike. Even if such symptoms are permanent, one has to be cautious in making the diagnosis "katatonic dementia." The operated case reported by the writer shows that on the basis of traumatic hystero-epilepsy symptom groups occur, very much resembling the later stages of katatonia.

3. *Ophthalmoplegia Interna Traumatica*.—Ernst Schultze reports a case in which an isolated paralysis of the ciliary muscle and sphincter iridis existed. Ophthalmoplegia interna is mostly found with tabes and paralysis caused by lues. But in this case it was most probably the result of a hemorrhage in the region of the oculomotor nucleus.

4. *The Trend of Investigation in the "Psychologic Studies."*—(To be continued).

(1903, No. 2, February 15.)

1. On the Psychophysiology of Negativism. RAGNAR VOGT.
2. Critical Remarks on H. Schmaus' "Lectures on the Pathological Anatomy of the Spinal Cord." F. NISSEL.
3. The Trend of Investigation in the "Psychologic Studies." W. WEYGANDT.
4. Recent Studies on Alcoholism. HOPPE.

1. *On the Psychophysiology of Negativism*.—According to Vogt negativism can be explained like the other katatonic symptoms by the supposition of a higher "persevering" power of the cortex. The perseverance of one motor impulse diminishes the susceptibility to other movements. In many cases the patients try to make a requested movement, but during this trial contrary impulses arise. If you ask a katatonic patient to shut his eyes, sometimes opening and closing movements vie with one another, until finally the eyes are suddenly closed.

Vogt believes that with every movement of a group of muscles the antagonists also become innervated. This condition of innervation of the muscles, and the intensified "perseverance" of the psychomotor phenomena in katatonics, explain negativism sufficiently. If now katatonic patients find often that they have great difficulty in making the required movements, they think such requirements a vexation and they become morose, thinking it perhaps unnecessary to try to fulfil the request. Thus negativism appears frequently to be wilful perversity.

2. *Critical Remarks on H. Schmaus' "Lectures on the Pathological Anatomy of the Spinal Cord."*—Nissel says that these lectures contain all that we know at present about pathological changes of the medulla spinalis. He thinks it important that the author is a pathological anatomist, while before him neurologists and psychiatrists have described the pathological anatomy of the nerve centers. This separation of one branch leads to one-sided opinions about it. Schmaus has tried to describe the anatomical changes in the spinal cord more correctly than it has been done before, from the anatomical standpoint. Nissel objects to the co-operation of a neurologist, to whom Schmaus committed the clinical part of his lectures. Then he alleges that Schmaus is in error regarding the lymphatic spaces. Nissel claims to have proven by experiments that the adventitious sheaths are the only true lymphatic spaces, and that the membrana adventitia of the vessels forms a biological margin between the ecto- and meso-dermic structures of the nervous system.

3. *The Trend of Investigation of "Psychologic Studies."*—Weygandt gives a full review or critique of the first volume of reports issued from the laboratories under the direction of Emil Kraepelin of Heidelberg.

4. *Recent Studies on Alcoholism*.—To be continued.

(1903, No. 3, March 15.)

1. On Hydrotherapy in Psychoses. W. ALTER (Leubus).
2. The Trend of Investigation in the "Psychologic Studies." W. WEYGANDT (Concluded).

3. Recent Studies on Alcoholism. HOPPE (Konigsberg).

1. "*On the Hydrotherapy of Psychoses.*"—The author considers hydrotherapy to be the best of all sedative measures. He has tried it in 79 per cent of his patients, not only on cases of furious excitement and grave acute insanity, but also on deeply depressed, hysterical and demented patients. He employed cure-baths of 1 to 6 hours' duration; prolonged baths, not less than twelve hours, and wet packs continued from one and one-half to twelve hours. The cure baths were given to sleepless patients, and to paranoiacs having outbreaks or exacerbations. The emotional manifestations of hebephrenic and katatonic patients could be largely controlled by cure-baths. For hysterics the wet packs surpassed the prolonged baths. In the remissions of paresis prolonged baths of gradually lowered temperature seemed to lengthen the period of remission.

Baths lasting more than twelve hours have been an absolutely effective remedy in all states of grave excitement; also in subduing hallucinations in dementia præcox, the prolonged baths have been very useful. The author did not in any case abandon the prolonged bath on account of a negative result. Repeatedly he continued it for very long periods. Occasionally it was continued six, eight, fourteen and twenty-three days. The author mentions a case of puerperal "exhaustion psychosis," in which a cure resulted from a bath of seventy-two hours. In mild hysterical cases and in convalescent patients who cannot sleep, Alter recommends wet packs: arteriosclerosis and heart disease do not contraindicate the measure. Hydrotherapy requires the strictest individualization to secure the benefits of it; but properly used, it permits a great reduction in the use of calmate drugs. Alter nevertheless gives opium to melancholics in increasing doses, supporting the medication by systematic wet packs which always hasten the effect of the drug. Generally all psychoses had a milder, more subacute course under the new treatment.

3. *Studies on Alcoholism.*—To be continued.

(1903, No. 4, April 15.)

1. On Paralyzes after the Employment of Creasote Phosphate. L. LOEWENFELD.

The writer believes that his observations upon this condition are unique. Creosote phosphate, under the name "Phosphot (e)," is put on the market by a manufacturing chemist as a remedy against tuberculosis. A colleague of Loewenfeld's tried the preparation in three cases of pulmonary tuberculosis in doses of one gram injected into the gluteal mass. Good results were observed, as regards the lung affection, after 15 or 16 doses; but in each case paralytic manifestations in the upper and lower extremities appeared. The manufacturers of the drug say that others have reported similar results. Loewenfeld gives a full description of two cases of this paralysis. The lower leg- and foot-muscles are first affected; the knee-jerk remains; sensory symptoms are slight; bladder and bowel are not affected. In one of the cases atrophy was present for some time in the peroneal region. The writer gives reasons for thinking that the paralysis is not altogether peripheral, and concludes that this, like most toxic paralyzes, is a neurone disease, the ventral gray horns and the peripheral motor nerves being affected simultaneously. The phosphoric acid part of the compound surely cannot be the cause of this palsy; and the creasote in it is only half of what is contained in "creasotal," so that Loewenfeld is at a loss to account for this untoward effect of creasote phosphate.

PICKETT (Philadelphia).

JOURNAL OF MENTAL SCIENCE

(Vol. 49, 1903, No. 1, January.)

1. Some Features in the Intimate Structure of the Human Cerebral Cortex. JOHN TURNER.
2. Insanity in Imbeciles. A. F. TREDGOLD.
3. The Training of Nurses in Institutions for the Insane. BEDFORD PIERCE.
4. Case Taking in Large Asylums. D. F. RAMBAUT.
5. Treatment of Phthisis in Asylums by Urea and its Salts. J. L. BUSKIN.
6. The Alkalinity of the Blood in Mental Disease. ROBERT PUGH.
7. The Abnormalities of the Palate as Stigmata of Degeneracy. E. H. HARRISSON.
8. Insanity from Hasheesh. JOHN WARNOCK.

1. *Structure of the Human Cortex*.—These new features described by Dr. Turner are (1) A beaded net-work which involves pyramidal cells of the cortex cerebri and which have not, hitherto, been observed in the human brain, but only around the nerve cells of some of the lower animals (guinea pigs and rabbits) when subjected to the influence of methylene blue injected into the tissues during life; and (2) an intercellular plexus of extremely fine fibrils which has, he believes, never before been actually demonstrated in any brain, human or otherwise.

The technic employed by the author is a modified methylene blue method, as described in the autumn number of *Brain*, 1900. The pericellular network which he has first described for human brain consists in mass of fine dark fibrils, on which, at varying distances, are small dark beads, or sometimes rings, which, as a rule, are the nodal points of the meshes. The size of the mesh and the coarseness of its fibrils also differ, so that sometimes one meets with a big-meshed net, having relatively few beads and with very delicate fibrils, at others the beads are larger, very closely clustered together or even partially coalescing and the fibrils much coarser. So far as he has yet been able to determine, this network is met with only within the cells of the pyramidal type. It could be seen over these in all the layers where they are met with except the skin layer and probably the innermost or lower of the spindle-cells. This network he believes to be of nerve structure, as it is found along the dendrites, and this is evidence, he believes, that these parts themselves are concerned in the conduction of nervous impulses. On the question of thorns the author believed these to be artefacts. The paper is well illustrated and is one of much suggestive interest, particularly as the author takes up a number of related physiological questions.

2. *Insanity in Imbeciles*.—Insanity is rarely met with in the more pronounced grades of imbecility. In the middle grades of imbecility, however, such a complication is far from being infrequent, and out of over 200 imbeciles whom the author has examined in the asylums of the London County Council, more than one-half at one time or another had been insane. The subject has not been discussed at all in England, and it deserves more attention. It is found, he thinks, that those imbeciles who subsequently became insane have for some years before the actual outbreak been prone to sudden fits or irritability, bad temper, moroseness or sulkiness often accompanied by acts of violence; or that they have been in the habit of wandering away from home, or that they have evinced a restless disposition. This mental instability appears to the author to be by far the most important factor in the causation of insanity in these patients. Uncontrollable ideation, with resultant delusions, uncontrolled emotional action are two very characteristic features in these imbeciles. They have emotional storms which are very common.

As for clinical types of insanity, the author says these are mania or

melancholia, mania being the most common form occurring in about 55 per cent of all cases: melancholia (probably the older definition of melancholia here being meant) in 40 to 45 per cent. Delusional insanity is very rare and general paresis occurred in only two or three per cent.

3. *The Training of Nurses*.—This is a short paper on the general topic of administration that contains very little that can be abstracted. The author in general praises the methods that are practised in our own institutions, and speaks of them as being adaptations of general hospital methods to asylum life.

4. *Case Taking*.—A short note on how to keep case books.

5. *Treatment of Phthisis by Urea*.—This is a short paper on the action of urea in the treatment of tuberculosis, in addition to some remarks about infection and disinfection in asylums. The physiological chemistry quoted in the paper is very fragmentary and belongs to an earlier generation. As to the exact value of urea in tuberculosis the latest researches do not bear out Harper's extravagant claims.

6. *Alkalinity of the Blood in Mental Disease*.—R. Pugh pursues his method for determination of the alkalinity of the blood as already spoken of in our abstract of his article on alkalinity of the blood in epilepsy in Brain already presented, q. v. He summarizes his work on epilepsy in the present paper and discusses the blood conditions in general paresis, mania and in melancholia. His conclusions are as follows: (1) The alkalinity of the blood is physiological in chronic mania, melancholia and dementia. (2) It is lowered in cases of mania, during the period of excitement. (3) It undergoes marked variations in epilepsy, e. g.: a. It is below normal during the inter-paroxysmal period; b. it undergoes a sudden and pronounced fall immediately prior to the onset of the fit; c. it undergoes a further diminution after the fit is over. This after-diminution depends upon the length of time, the severity of the muscular spasms, and the degree of the alkalinity in the inter-paroxysmal period; d. there is a gradual return of the blood to its normal alkalinity, which takes place in five to six hours; e. there is a relationship between the degree of the alkalinity and the onset of fits, e. g., the higher the alkalinity the less liable is the patient to have a fit; f. it is impossible to elevate and maintain the alkalinity within physiological limits for any appreciable length of time by the administration of drugs. (4) It undergoes a diminution in dementia paralytica. This diminution is constant and well marked, and is probably due to the products of degeneration in the circulation. The variations in the diminution met with depend upon the type, progress and duration of the disease.

7. *Abnormalities of the Palate*.—The author first gives a summary of the various points of view of others, and the relationship between types of palate and mental qualities and describes twelve different kinds of palates and comes to the amazing inference: The palate of insane heredity is essentially a palate which is shallow or, at any rate, of the average depth in front, whatever its other characteristics may be. His general conclusions are summed up as follows: (1) Abnormalities of the palate are common in the insane; (2) these abnormalities may be roughly classified into two large groups, of which the former contains the palate of the hereditary psychopath and the latter the palate of the general degenerate; (3) the former palate is variable in its general type, but as a whole is shallow, or, at any rate, of the average depth in front; (4) the latter palate is also variable in its general type, but is in the main characterized by an increased depth at the level of first bicuspid.

The general criticism that may be offered is that the author makes generalizations from the few cases studied rather than from a general survey of the whole field.

8. *Insanity from Hasheesh*.—The author's experience in Egypt as di-

rector of the Egyptian Hospital for the Insane at Cairo puts him in a position to speak authoritatively on this interesting topic. Large quantities of cannabis are used by the inhabitants of the Egyptian towns, where the drug is mostly consumed by smoking in the gozeh and in cigarettes. Hash-eesh users usually degenerate morally, and for this reason, if for no other, the habit is disapproved of in Egypt. Insanity from hasheesh belongs to the toxic group and in many regards is similar to that caused by opium, alcohol, cocaine, etc. The author compares the insanity from hasheesh along lines known for alcoholic intoxication. Thus he describes (1) Temporary intoxication, in which the smoker becomes dull and drowsy, feels pleasantly exalted and is at peace with the world. This is the common state to be observed among habitués of hasheesh cafes. There is very frequently a staggering gait, and if the hasheesh smoker is annoyed or interfered with he is liable to become excited, irritated and loses control. The author contrasts hasheesh, opium and alcohol by saying that the mental pose of the hasheesh smoker is more subjective than alcohol and less so than that of the opium user. The alcoholic is the most objective and demonstrative of the three. (2) Delirium from hasheesh, which is usually accompanied by hallucinations of sight, hearing, taste and smell. There is great exaltation, much restlessness and sleeplessness with motor phenomena of staggering, although there is no tremor or ataxia. Physical excitation and gastro-intestinal disorders do not occur. (3) Mania varies in degree of acuteness from a mild, short attack of excitement to a long, furious mania, ending in death. (4) Chronic mania may result. (5) Chronic dementia usually is a final stage of the preceding forms. (6) Cannabinomania is employed by the author to describe the condition of many hasheesh users between the attacks of the other forms. The user is usually a good-for-nothing, lazy fellow, who lives by begging and stealing, and pesters relatives for money to buy hasheesh.

The author gives some statistics bearing on the amount used, and a number of interesting and important legal suggestions. He believes that hasheesh is a much more dangerous drug in Egypt than India.

(Vol. 49, 1903, No. 2, April.)

1. Bacteriological and Clinical Observations on the Blood of Cases Suffering from Acute Continuous Mania. L. C. BRUCE.
2. The Case of an Unrecognized Degenerate Punished by the Law. E. GOODALL.
3. Nomenclature of Mental Diseases. A. R. URKUHART.
4. The Care and Treatment of Persons of Unsound Mind in Private Houses and Nursing Homes. E. W. WHITE.
5. Lunacy and the Law. T. O. WOOD.
6. Note on a New Case Book Form. W. R. DAWSON.
7. Notes on Hallucinations, II. CONOLLY NORMAN.

1. *The Blood of Acute Continuous Mania*.—L. C. Bruce of Murthly contributes a series of observations on the blood in acute mania. A few years ago he made some observations on the blood in insanity and was unable to find any micro-organisms and he devised a new method of making aseptic necrotic areas subcutaneously and then subsequently studying this area to ascertain the bacteria found therein. Acting on this theory he took a case of acute mania, an adult woman, and with anti-septic precautions injected into the soft tissues of the flank two c.c. of turpentine. An abscess formed and on the third day after the injection he aspirated some fluid and obtained a small culture of a small diplobacillus. Since this time he has made 24 similar observations and has observed the diplobacillus in seven instances. It has been found only in

cases of acute continuous mania, although other forms were studied. The details of the cultural characters are given. In a further portion of his paper he gives the results of the study of the leucocytes in fifty cases of acute insanity. Adopting 6,000 to 10,000 leucocytes per cubic mm. as his normal, he has studied leucocytosis in those who became chronic and those experimented on as previously reported and recovered, and in those in whom abscess was made in a chronic case which did not recover. In recent cases he finds that there is a high leucocytosis, thus bearing out the later contention that acute manias are always of infectious origin. This leucocytosis is mostly in the polymorphonuclear cells. In the second class of cases the leucocytosis tends to remain between 12 or 60 and 16,000, but the polymorphonuclear cells are rarely over 50 per cent. In his third group of cases leucocytosis was high, but gradually sank and recovery took place, mental improvement, the author declaring to be in proportion to the leucocytosis. His general conclusions are as follows: (1) If these blood observations are correct, they practically prove that acute continuous mania is an acute infective condition, and that when recovery takes place a condition of immunity is established. (2) They prove that, although the patient apparently recovers, the disease remains latent; hence the persistent leucocytosis, a point which might be of great importance in life-insurance examinations; (3) an examination of the blood is a valuable aid to prognosis, as for instance: If a case of mania has lasted for a month and remains maniacal and sleepless. The blood examination gives a leucocytosis of 14,000 per c.mm. of blood, with a percentage of 60 or below 60 of the multinucleated cells. The chances of an immediate or early recovery are poor. On the other hand, if the blood examination gives a leucocytosis of 18,000 or 20,000, with the multinucleated cells in a percentage of 70 or above 70, the prognosis is good. It is well when examining the condition of the blood to aid prognosis, to examine the blood on at least two consecutive days.

2. *Degenerate Punished by the Law.*—The case is that of a man, thirty-five years of age, who showed, on transferral to an asylum from a prison to which he had been condemned for twelve months' hard labor, symptoms of congenital deficiency with depressions, visual and aural hallucinations and agitation. The patient's ancestry was bad, and he had been in jail nine times previously. Anthropological measurements are also given in the paper. The author's conclusion is that the type of punishment meted, i. e., that of short-term imprisonments and discharge for this class of cases is useless and injurious to the individual and unjust to society. The prison is not a proper place for this type of criminal. He is mentally diseased and should be shut up for life in an asylum or reformatory.

3. *Nomenclature of Mental Disease.* The author gives some suggestions on the classification of the insane in which there is very little new and nothing striking or noteworthy.

4. *Care of Insane in Private Houses and Homes.*—The paper the author considers the natural outcome of Sir William Gowers' recent address on the care of the insane. It takes up the advantages of certified single care, and that they are privacy, domesticity, visits of friends, avoidance of the stigma of treatment in a lunatic asylum; whereas the disadvantages are the absence of skilled medical treatment, unskilled nursing, monotony, insufficient moral control, interference of friends, limited supervision, want of tact and business capacity on the part of the custodian. These are the advantages or disadvantages in the care and treatment of the duly certified person of unsound mind in private houses. As to the taking charge of a person of unsound mind not under service in a private house or nursing home the author's advantages and disadvantages are much the same, the want of official supervision being perhaps the most

serious of all the disadvantages. There are a number of useful suggestions in the paper, but they do not have very ready usage here.

5. *Lunacy and the Law*.—T. O. Wood discusses several features in the English Lunacy Law, which are of secondary interest to those practicing under different regulations.

6. *Note on a New Case Book Form*.—This is a short note on the method of keeping clinical histories and has no particular points one way or another.

7. *Hallucinations*.—The author here gives the histories of thirteen patients suffering from different forms of hallucinations. The paper is too detailed to admit of ready abstraction, but it contains many interesting suggestions, bearing more particularly upon the theory of psycho-motor verbal hallucinations, whereas he does not discuss any of this type. Those that are given show interesting connections with the forms described by Seglas.

8. *Clinical Notes and Cases*.—This number also contains Notes on some Pathological Cases, by M. J. Nolan, the swallowing of foreign bodies, such as nails, bristles, etc., by demented; a case of suppurative cholangitis; melancholia with universal acute eczema; senile melancholia associated with aneurisms of the aorta and rupture, and also a few Cases of Abdominal Surgery in the Insane, by Robert Jones; A Case of Hebephrenia, by W. R. Dawson; a Case of Thoracic Aneurism in a Secondary Dement, Simulating Mediastinal Growth, by Robt. Pugh; and Status Epilepticus Complicated with Scarlet Fever, by G. W. Greene. JELLIFFE.

BRAIN

(Vol. 25, 1902, Winter, No. 4.)

1. Some Observations on the Primary Degeneration of the Motor Tract. F. W. MOTT and A. F. TREDGOLD.
2. On the Pallio-tectal or Cortico-mesencephalic System of Fibers. CHARLES E. BEEVOR and SIR VICTOR HORSLEY.
3. A Case of Arrested Development of the Cerebellum and its Peduncles; with Spina-Bifida and Other Developmental Peculiarities in the Cord. W. B. WARRINGTON and K. MONSARRAT.
4. Toxic Degeneration of the Lower Neurones, Simulating Periphery Neuritis. STANLEY BARNES.
5. On Certain Blood Changes in Idiopathic Epilepsy. ROBERT PUGH.

1. *Primary Degeneration of the Motor Tract*. The authors describe the clinical course and microscopical appearances in four cases: two amyotrophic lateral sclerosis, one of progressive muscular atrophy and one of chronic rheumatoid arthritis with muscular atrophy. They say that degeneration of the motor tract is the anatomical lesion present in severe diseases having distinct clinical features. Thus it occurs in Friedreich's disease, in combined lateral and posterior sclerosis and in general paralysis, but in these other neurone systems are also affected. Degeneration may also result from local lesions as in the anterior poliomyelitis, polioencephalitis, embolus, etc. And lastly, cases exist in which not a degeneration but an arrest of the development of the motor tract occurs, apparently due to simple agenesis. It is with the primary degenerations, however, that occur in amyotrophic lateral sclerosis, progressive muscular atrophy, bulbar paralysis or primary spastic paraplegia that the paper deals.

Speaking of the nature of the morbid processes they believe that the changes in these cases are those of a sub-acute or chronic degeneration of a portion or the whole of the motor tract, that is of the upper segment alone, of the lower alone, or of both segments together. The cases they describe demonstrate that the change is a slow attack of individual neu-

rones. The cells become shrunken and irregular in outline, the Nissl bodies gradually disappear, their space is taken by a large collection of yellow pigment, the nucleus becomes smaller and its outline displaced. Later it is displaced to the periphery of the cell and finally disappears. There is true degeneration in the nerve fibers and in the end organs. Neuroglia gradually takes the place of the atrophied nerve fibers causing a secondary sclerosis. The authors believe that this progressive degeneration is due to the fact that the neurone has reached the stage at which its inherited vitality is exhausted and that this presents a probable indication of presenile decay, but they present no evidence one way or another.

On the question of the pathological relations between the upper and lower motor segments they say that while there can be no doubt of the existence of trophic action between the upper and lower parts of the efferent tract, in some cases the presence of degeneration in one portion resulting from degeneration in the other seems to be rather the exception than the rule, and they consider that it is now well established that disease may involve either segment separately or both together, and if each segment of the motor tract is specially susceptible to the influence of particular poisons it seems legitimate to conclude that there may be different hereditary tendencies in the two tracts, in some persons there may be a premature tendency to decay in the upper portion, in others in the lower portion, and in still others throughout the entire motor system. Other neuronc involvement was present in some of the cases. These were considered secondary by the authors and were discussed under two heads, association fibers and efferent fibers.

2. *Pallio-tectal System of Fibers.*—An investigation of fibers composing the crus cerebri in a number of animals, 20 cats, 11 monkeys, 1 dog and 2 badgers. Local lesions in the cortex had been made by Horsley. While tracing the degenerations which resulted from such lesions the authors especially followed out the connections which have for some years been known to exist between the cortex and the mid-brain. The Bush modifications of the Marchi method were used and serial sections studied. The results obtained are very detailed and the original should be consulted, but the general statement may here be made that as the research was founded chiefly on observations on the cat, representing the carnivora, and the macaque monkey, representing the primates, the results are to be understood as applicable only to the higher of these animals in which there is a well-developed non-excitabile area (frontal) in front of the so-called motor or excitabile cortex. The general scheme of the pallio-tectal system is stated by the authors to be as follows: From the frontal cortex no fibers can be traced to the mesencephalon; from the temporo-sphenoidal cortex very few fibers; while from the occipital lobe a fair number, and from the excitabile cortex a large number of fibers can be followed to the corpora quadrigemina and mesencephalon. The course taken by the different group of fibers is then traced by the authors. We can not give the details in this place, but refer the reader to the original.

3. *Arrested Development in the Cerebellum.*—The authors describe in full the clinical history of a case of restricted development of the cerebellum, and then describe the macroscopical appearances of the brain and cord; the histology of the medulla, cerebellum, pons and membranes with the following results. A child, aged six weeks, presenting a lumbar spina bifida ruptured at or before birth, and now healed; hydrocephalus, paraplegia of both lower limbs. Death from intercurrent gastro-enteritis.

(1) In the brain. Arrested development of the cerebellum and of its efferent and afferent fibers, and of their nuclei of origin; of the restiform bodies, the olives, accessory olives, external arcuate fibers and nuclei arcuati, middle peduncles and transverse fibers of the pons, pontal nuclei, vestibulo-cerebellar tracts, superior peduncles, gray nuclei of cerebellum, red nuclei of tegmentum; possibly the cerebro-pontal tracts.

(2) In the cord. General attenuation of this structure throughout, division at the level of the fourth dorsal vertebra into two equal halves; marked enlargement of the central canal which divides with the division of the cord; the presence of various irregular cavities in the cord and disintegration of its central parts in the lower dorsal region; normal development of the gray matter with well-formed ganglion cells; scanty development of white matter, its place being taken by tissue of neuroglial structure.

(3) The coverings of the cord. The whole cord as far as the upper cervical area, embedded in a fine connective tissue with large vascular channels; the laminae and spines of the fourth and fifth lumbar vertebrae deficient; an exostosis dividing the canal at the level of the fourth dorsal vertebra. In an appendix they give a complete bibliographical summary of cerebellar atrophies arranged on the basis of their proper pathogeny.

4. *Toxic Degeneration of the Lower Neurones*.—Dr. Barnes reports the histories of seven cases in which the clinical picture resembles in many respects that of peripheral neuritis and yet in other ways corresponds to the type of progressive muscular atrophy. The clinical histories are given very thoroughly, and his conclusions are very interesting. These may be summarized as follows: (1) There is a clinical type which is usually the sequel of acute specific fevers, which resembles the paralysis seen in multiple neuritis, but which is associated with great atrophy of the hand muscles. It usually begins about the second or third week after the febrile condition and involves the muscles from the periphery to the trunk to a varying extent. It may progress for a few days only, or for several months. Sensory signs are present, but slight in degree. Although the condition somewhat resembles progressive muscular atrophy, the prognosis and etiology of the two conditions are probably widely different from one another.

(2) After a certain stage, when once definite improvement has begun, relapses are not common, and there is a constant tendency to improvement. Even years after the subsidence of the acute condition considerable improvement may still take place, the small muscles of the hands being the last to recover. Contractures are rarely developed.

(3) Probably the condition is one of toxic degeneration of the lower neurones, the motor neurones being more particularly affected.

5. *Blood Changes in Idiopathic Epilepsy*. Dr. Robert Pugh of Claybury presents the results of the examination of forty cases of epilepsy in which differential counts of the leucocytes have been made, and the study of the alkalinity of the blood carried on. The author concludes that in epilepsy there are marked variations in the alkalinity of the blood, most of which manifest themselves by diminution of alkalinity. The average alkalinity during the (a) interparoxysmal period is lower than the average of the control cases. (b) There is a sudden pronounced fall immediately prior to the onset of the fit. (c) There is a certain diminution after the fit is over. Accounting for the interparoxysmal diminution the author suggests two hypotheses: (1) It may be explained by the gradual accumulation of toxins of an especial nature in the blood, and (2) that it may be the result of deficient metabolism, general metabolism being known to be below par in epilepsy. Accounting for the pronounced fall in the alkalinity immediately prior to the onset of the fit. The most likely origin of the acids the author claims are either (a) neuronic, (b) muscular. In the first case, the increased amount of acid being derived from the muscular and the glandular tissues. The author says that the exact rôle which the diminution in the alkalinity of the blood plays in the production of an epileptic attack is difficult to estimate. It is of utmost importance for any

cell in the body to have a proper adequate and pure supply of blood, and this applies especially to cerebral neurones, as they are extremely sensitive to a change in environment. The author believes that he has obtained good results in the treatment of epilepsy by maintaining the alkalinity of the blood within physiological limits, but unfortunately he says it is impossible to elevate and maintain the alkalinity by the administration of drugs.

With reference to the leucocytes, there is a distinct leucocytosis, particularly after the attack. The author's general conclusions are as follows: (1) The alkalinity of the blood in the interparoxysmal period is lower than the average of the control cases; (2) the diminution is gradual and progressive and is more marked in those cases suffering from gastric catarrh and constipation; (3) there is a marked sudden and pronounced fall immediately prior to the onset of the fit; (4) there is a further fall in the alkalinity after the fit is over; this diminution is seen from three to ten minutes after the attack; (5) this after-diminution depends upon the duration and severity of the muscular twitching, and upon the degree of the alkalinity in the interparoxysmal period; (6) there is a gradual return of the blood to its normal alkalinity, which takes place in five to six hours, the rise being more marked in the first hour.

JELLIFFE.

LE NEVRAXE

(Vol. IV. Fasc. 3. 1903.)

8. On the Form and Development of the Protoplasmic Prolongations of the Spinal Cells in the Higher Vertebrates. T. GEIER.
9. Researches on the Central Acoustic Tracts: The Bulbo Mesencephalic Acoustic Tracts. VAN GEHUCHTEN.
10. The Inhibitory Fibers of the Heart belong to the Pneumogastric Nerve and not the Spinal Nerve. VAN GEHUCHTEN.

8. *Form and Development of Protoplasmic Prolongations.*—Dr. Geier has been studying the dendrites of the spinal cells of the horn fibers, using kittens and rabbits for investigations. Golgi's method was used. He finds that the dendrites of the anterior horns are smooth in contour and differ from the posterior horns which have an irregular contour. The spinal dendrites extend in a straight line, while those in the posterior horn are irregular and sinuous. The dendrites in the cells of the anterior horns have few and insignificant collateral appendices while those of the posterior horns are quite complex. There are many variations in the dendrites of the new-born animal as contrasted with those of the adult, for full details of which the reader is referred to the original.

9. *The Upper Auditory Tract.*—Van Gehuchten here presents results of an extended series of experiments performed on rabbits and studied by the Marchi method. Five series of experiments are given which lead Van Gehuchten to different conclusions than those heretofore held. In the first group of cases he tore the seventh nerve out through the stylo-mastoid rim. In this way the ventral fibers of the trapezoid body were involved. Serial sections showed that degenerated fibers were found passing into the trapezoid body vertically above and below the actual level of the lesions. In the trapezoid these fibers could be traced to the opposite side; they turn upwards, those ventrad passing in front of and those dorsad behind the trapezoid nucleus. These two degenerated bundles so united to form a compact strand of fibers which lay in contact with the lateral aspect of the inferior nucleus of the lateral lemniscus. Tracing this strand of fibers upward it was found to turn outward and then backwards finally entering and forming the more superficial part of the lat-

eral fillet. From this place onward the fibers could be traced to the posterior quadrigemina where they terminated close to the superior nucleus of the lateral lemniscus. In the second series of cases, similar degenerations were found. In a third series a lesion was made by cutting downward and outward through the floor of the fourth ventricle, dividing both the dorsad and ventrad fibers of the corpus trapezoid. Similar conclusions as those already outlined were reached by this method of investigation. The fourth series of experiments was made to ascertain the cells of origin of the two strands of the corpus trapezoid tract. If a lesion was restricted entirely to the accessory nucleus of the eighth nerve degenerations corresponding to those just described resulted. On the question of the relation of the medullary striæ in the secondary auditory path the author found that attempts to cut these on the floor of the fourth ventricle were unsuccessful but they were found destroyed in an experimented animal in which the seventh nerve had been torn out and had ruptured the acoustic striæ. Degenerated fibers can be followed from the point of injury to their raphe where they crossed dorsad to the upper fibers and the trapezoid body until they became posterior to the olivary nuclei. They then turned upwards, lying behind the dorsal trapezoid tract and internal and ventrad to the motor fifth nucleus. They entered the lateral lemniscus at the upper border of the upper cerebral peduncle, being mingled with the fibers of the dorsal strand of the corpus trapezoid. They finally ended in the nucleus of the posterior quadrigemina itself, thus tracing the fibers of the secondary auditory path higher than those previously described. The author was unable to verify the opinion of Held that neither the dorsal nor ventral auditory paths could be traced to the inferior corpora quadrigemina or cortex. Thus the author holds that the central auditory path is in reality a bulbo mesencephalic rather than a bulbo-cortical one. The illustrations are numerous, there being over 62 illustrations accompanying this extremely interesting and suggestive article.

10. *Inhibitory Fibers of the Heart*.—Van Gehuchten controverts the old idea that the inhibitory fibers of the heart arise in the spinal accessory and pass to join the vagus by its internal branch.

He concludes that (1) the inhibitory fibers of the heart do not come from the spinal accessory nerve but are derived from the vagus itself. (2) His investigations on the innervation of the larynx (see abstract of last number) go to prove that all of the bulbar fibers of the spinal accessory are distributed to the muscles of the larynx; the eleventh nerve has an exclusive medullary origin and (4) the vagus gives off a superior and inferior system of fibers exclusively motor, including motor fibers destined to all the muscles of the larynx exclusive of the crico-thyroid, which is supplied by the inferior laryngeal nerve and a secondary series of inhibitory fibers to the heart.

JELLIFFE.

REVUE DE PSYCHIATRIE ET DE PSYCHOLOGIE EXPERIMENTALE

(January, 1903.)

1. Tests in Psychopathology. ED. TOULOUSE and N. PIÉRON.
2. Experimental Researches on the Psychology of Memories. N. VASCHIDE and CL. VURPAS.
3. Study of Morbid Obsessions.

1. *Tests in Psychopathology*.—The aim of this article is to indicate the lines along which the development of psychopathological tests must proceed if they are to be more exact and comparable. In their introduction the authors critically review the work of Guicciardi and Ferrari and of Tamburini, Badaloni and Brugia. They then proceed to a brief statement

of their own methods illustrated by their application in the domain of memory. They believe in the general principle that the methods for the study of the abnormal mind should be the same as those used for the study of the normal, for the abnormal mind differs only from the normal in showing exaggeration of certain characters or defect of others—differences only of more or less. Applied to an experimental study of the memory of sensations their method would involve the use of a definite multiple of the previously determined minimum perceptible stimulus in the sensory field under consideration. In this way only can results comparable as between different subjects be obtained as no stimulus is felt the same by all. There is no absolute in psychology, only the relative. In the study of the memory of perceptions the optimum or quantity best perceived is used as a basis, while in the study of the memory of ideas sentences constructed of identical length, in the same style, containing the same number of ideas disposed in the same way are used, thus detaching as far as possible the idea from its setting. The article closes with a few words on the calculation of errors.

2. *Psychology of Memories* (continued).

3. *Morbid Obsessions*.—The authors have studied for this article in all ninety-seven cases. They have studied only those cases in which the obsession was the essential psychopathic symptom, not differentiating for their purposes the phobias from the morbid obsessions. The results of their studies are as follows:

Of the 97 cases examined 76 were men and 21 women. The occupations were distributed as follows—8 clergymen, 11 merchants, 11 office employees (all men), 14 domestics (all women), 17 artisans, and 29 persons in different intellectual professions. The greater part of the obsessed were thus from intellectual pursuits; 92 per cent of all cases showed hereditary tendencies. Of 94 cases 7 only showed no afflicted relatives, 38 showed 1 or 2, 30 cases had 3 to 5, and 19 cases 6 or more. Thus in the great majority of cases at least 3 members of the family had been afflicted. Of 88 cases 7 showed no heredity, in 32 heredity existed on one side only, and in 49 on both sides. One-third of all cases showed a homogeneous heredity. A study of the mental life of these cases shows them to have been possessed of a peculiar character from early life, marked by indecision, irritability, desire for solitude, painful and disagreeable emotions, egotism and often hypochondriacal ideas. In 77 of the cases the phenomena manifested themselves at twenty years of age, in 11 cases after twenty years. The development was usually progressive and by insensible degrees. This progress, however, may be interrupted by exacerbations, especially as a result of influenza, menstrual difficulties, puberty, etc. Occasionally as an accompaniment of the obsession is found epileptoid, hysteriform, vertiginous or syncopal attacks, and sometimes complicating this condition sexual perversion, sexual excitability (4 cases), masturbation (42 cases), or other sexual anomalies (28 cases). Occasionally these cases dement, and then arises the question of differential diagnosis between pure morbid obsessions and dementia præcox with obsessions.

(February, 1903.)

1. The Mental State in Cerebral Tumors. A. VIGOUROUX.

2. Experimental Researches on the Psychology of Memories. N. VASCHIDE and CL. VURPAS.

1. *Mental State in Cerebral Tumors*.—The author deals with these three points in this article; first: have these psychic troubles characters which point to the presence of a tumor? second: are they of such a nature as to indicate its location? third: can they be classified? After a careful review of the opinions of writers on this subject the authors answer these queries negatively. Psychic symptoms may or may not accompany a tumor in a given location, and their nature is independent of the nature of the tumor as such and may be the result of accidental conditions, such as inter-

ference of circulation, meningitis, the impregnation of the brain by toxic products of disassimilation, surrounding inflammation and many other causes. Finally the author concludes, with Ball, that when a true psychosis is found associated with a cerebral tumor, there is every reason to believe that the association is a simple coincidence.

2. *Psychology of Memories* (concluded).—An experimental study confined solely to the domain of psychology and having little interest from the standpoint of psychiatry.

WM. A. WHITE (Binghamton, N. Y.)

MISCELLANY

HYPNOTICS AND THEIR DANGERS. Dr. Nestor Tirard (*Lancet*, April 11, 1903).

In a treatise on some dangers of hypnotics the author draws the following conclusions: He says that he is convinced that in the employment and in the selection of a hypnotic sleeplessness is perhaps the least important factor to be considered, although it may be the sole symptom of which complaint is made. The cause must be ascertained, together with any indications of idiosyncrasy. The co-existence of other diseases must influence the selection of the hypnotic, and even when, for the individual case, a suitable hypnotic has been found, risks of repeating moderate doses must be borne in mind. Generally the dose has to be gradually increased, especially when the cause has not been ascertained or satisfactorily dealt with, but with some drugs, e.g., sulphonal, trional and tetronal, the danger appears to lie in the total quantity taken, even though each dose may have been small. But perhaps the greatest danger of all is incurred when the patient is given a prescription for a hypnotic, and this danger has certain analogies with the medicinal employment of alcohol. Many of us believe that at the height of fever or in conditions of collapse, alcohol is often necessary, but it is never recommended to our patients for indefinite use, still less for increasing doses. Armed with a prescription for a hypnotic patients commonly go on taking the drug long after the relief of the special circumstance for which it was prescribed, and the prescription may be used to facilitate the acquisition of large quantities of a dangerous drug obtained, perhaps, at several establishments in small amounts. Notwithstanding the relief which many have derived from the use of hypnotics, he is in favor of any step which tends to prevent their administration, except by the medical man or under his immediate directions. He has seen so much of the dangers of hypnotics that he would prohibit their sale to the public or would allow them only to be dispensed to a freshly-dated prescription, which should be retained by the druggist or returned to the prescriber.

JELLIFFE.

NERVE SUTURE AND NERVE DEGENERATION. P. B. Henriksen (*Lancet*, April 11, 1903).

In a study on the subject of nerve suture and nerve degeneration, the author says, considering the question of whether a degenerated nerve can be supposed to be to some extent conducting: This question may be solved in two ways—either by proving conducting elements in the degenerated nerve or by proving effects of the nerve function during the degeneration period. According to what has formerly been mentioned, the new fibers must be supposed to be formed of elements in the peripheral part of the nerve. What are these elements, and at what time can they be shown as continuing courses fit for conducting a nerve impulse? When a nerve is divided the weight of a corresponding muscle will diminish rapidly. By weighing the muscle twelve days after dividing one peroneal nerve of a rabbit it was found that the muscle corresponding to the divided nerve weighed 4.77 grams, while the muscle on the other side weighed 6.98

grams—a difference of 33 per cent. When the nerve is united again motility comes earlier than the electrical reaction. In several of the experiments the difference seemed to be considerable, as the animals could use the operated limb as well as the other limb long before electrical reaction was observed. If the muscles have thus resumed their activity, and in consequence of a return of nerve conduction, one may suppose that this also may be shown by changes in the microscopical appearance of the muscles or in the cessation of atrophy or by increasing weight. To examine these circumstances, the third series of experiments was made in the following way: The peroneal nerve was divided on both sides. On the one side one of the ends was pulled out through the slit in the fascia and fixed subcutaneously, while the slit for the rest was closed with catgut. In this way the nerve was prevented from uniting. On the other side the divided nerve was permitted to unite. The animals were killed after some days' interval, and both the united nerve and the central and peripheral ends of the non-united nerve were hardened for microscopical examination. The muscles supplied by the peroneal nerve were carefully dissected out as similarly as possible on both sides and were then immediately weighed. A piece of the muscles was cut out from corresponding places of the muscles on both sides and hardened in formalin for microscopical examination.

JELLIFFE.

MASSAGE IN LOCOMOTOR ATAXIA. Dr. de Frumerie (*Le Progrès Médical*, Feb. 28, 1903).

In every manual treatment it is necessary first to determine whether the effect desired is stimulation or sedation. In tabes the author confines his massage to sedative measures, avoiding any brusque or energetic movements which so often lead to hyperesthesia. The suitable types of manipulation for this disease are superficial or deep effleurage, vibratory motion of the abdominal viscera, and prolonged compression, the last sometimes overcoming the lightning pains when energetic measures have had no effect. For paralysis of the bladder the organ should be emptied of its infected urine and one-third filled with boric acid solution; then the curved fingers are pressed down behind the pubes and gentle tremulous motion set up. The perineal region may be subjected to effleurage and continuous pressure. When there are contractures, one must avoid the contracted muscles, the flexors, and by every means possible try to strengthen their antagonists, the extensors. The flexors then should be slowly and cautiously stretched by passive motion. Where there are no special contractures, a general stretching or extension may be brought about as follows: The patient lies on a bed and places the feet against the chest of the operator; then, without bending his knees, he is seized by the hands and pulled forward, the operator at the same time giving him a tremulous movement. This affects the spinal cord, the sciatics, the ligamentous and muscular tissues of the back, and the brachial plexus, without fear of injury to the femoral neck, which is so fragile in certain tabetics.

BASTEDO.

MYASTHENIA GRAVIS. Charles S. Myers (*Journal of Pathology and Bacteriology*, Sept., 1902).

Three signs may be described as pathognomonic of myasthenia gravis. They are (1) ready fatigue of certain or general voluntary movements either to a succession of tetanizing currents applied to the nerve or to volitional impulses descending from the brain. (2) The exacerbations and remissions shown in the course of the disease, and (3) the tendency to a fatal implication of the muscles innervated by the bulb. To these must be added other very important general signs mainly of a negative character. (4) There is no reaction of degeneration; the muscles react to a faradic current of normal intensity, or more often require one somewhat stronger

than normal. (5) The muscles, as a rule, show neither atrophy nor fibrillar contraction. (6) Sensation and intelligence are unimpaired. (7) The reflexes may be normal, are often feeble, but perhaps most frequently are somewhat exaggerated. Clonus is absent. (8) Purely voluntary muscles are alone affected. The mechanisms of defecation and micturition are never impaired. (9) The myasthenia, although often unequally marked on the two sides, is almost invariably bilateral. Myasthenia gravis is distinguished from hysteria by absence of stigmata or disturbance of sensation; from clonic bulbar paralysis by the extraordinary remission and exacerbation of symptoms, the general absence of muscular atrophy, of fibrillary contractions and degenerative electrical variations. Of 22 cases on which autopsies were performed, 8 showed no pathological condition of the nervous system. In 3 cases minute aneurisms or vascular dilations were noted; and in 2 the cerebral arteries appeared thickened. Eight cases examined by Nissl's method, in 3 slight chromatolysis was found. Hemorrhages in the central nervous system always recent and frequently numerous were found in 6 cases. The peripheral nerves and muscle fibers were examined several times, yielding varying changes in the muscles and rarely changes in the nerves. The writer does not believe that either the muscles or the higher parts of the brain are primarily affected. The disease probably arises from some autogenetic-toxine. The end plate is affected and with it the entire neurone, secondarily.

W. B. NOYES.

THE DIAGNOSIS OF BRAIN ABSCESES. Herman H. Hoppe, M.D. (Journal American Medical Association, March 14, 1903).

Given a certain group of signs and symptoms which lead us to suspect the presence of a brain abscess, the first point is tracing the source of infection. Bergman says that without the *Streptococcus pyogenes aureus* there cannot be a brain abscess. But to find the external source of infection is difficult. Bergmann divides brain abscesses into four groups. (1) Traumatic abscess caused by an open scalp wound or a fractured skull. (2) Those abscesses resulting from a purulent inflammation of bone or bony cavities, (a) otitic abscess, (b) rhinogenetic abscesses, (c) abscesses due to caries of the bones of the skull. (3) Tuberculous abscesses. (4) Metastatic abscesses. The symptoms of brain abscess may be grouped as follows: (1) Symptoms due to deep-seated suppuration, (2) symptoms due to intracranial pressure, (3) focal symptoms. Fever and other signs of sepsis, chills, localized pain and tenderness, vomiting and, at times, distinct mental symptoms are important symptoms. Optic neuritis is more frequent than in brain tumor, but not so well developed. If it is absent the abscess is either very acute or small. It is most marked in abscess of the frontal lobe. Focal symptoms help much to make the diagnosis if the abscess is present in the psychomotor area, little if present in the occipital, temporal or frontal lobes. The closer the abscess to gray matter, and the greater the destruction of tissue, the more focal signs are developed. A large abscess of the centrum ovale may exist without focal signs.

W. B. NOYES.

Book Reviews.

A TREATISE ON HUMAN ANATOMY IN ITS APPLICATION TO THE PRACTICE OF MEDICINE AND SURGERY. By JOHN B. DEEVER, Surgeon in Chief to the German Hospital, Philadelphia. In three volumes, illustrated by 499 plates nearly all drawn for this work from original dissections. Vol. III. Abdomen, Pelvic Cavity, Lymphatics of the Abdomen and Pelvis, Thorax and Lower Extremity. P. Blakiston's Son & Co., Philadelphia.

The arrival of the third volume of Deaver's Surgical Anatomy has been anxiously awaited by all possessors of the first two volumes, and their fondest anticipations have been fully realized upon their receipt of the final volume which has more than kept up the high standard already established by the first two.

The excellence of the plates from an artistic standpoint and their correctness as to anatomical detail, can hardly be surpassed. In the text the author has not gone into the minute anatomical description of the different organs, yet he has indicated most of the surgical procedures which may be required and has presented it in a clear and concise manner.

Volume III takes up the Abdomen, Thorax and Lower Extremity. The surface anatomy of the abdomen and thorax is beautifully depicted with tracings of the main vessels and contained organs, giving the anterior, posterior and lateral views; then follow descriptions of the superficial and deep fasciæ and the muscles. The anatomy of the inguinal canal is well brought out and is made perfectly clear by the numerous illustrations. The peritoneum, the reflections of which the student is apt to find a stumbling block, is made perfectly plain by means of four full page plates. The abdominal and thoracic organs are all described and beautifully depicted, particularly the heart and lungs. The lower extremity has its surface anatomy marked out and particular attention is given to the anatomy of Scarpa's triangle, the gluteal region and femoral hernia, also the popliteal space. The joints of the lower extremity are fully described, and the deformities commonly met with, as well as the surgical procedures indicated for their relief. Dislocations are also touched upon as well as excisions, the lines of incision for which are indicated not only for the joints themselves but for the bones of the foot. Fractures are lightly gone over, likewise amputations, ligation of arteries and nerve stretching.

The author has left little ground for criticism in this work, and the only one we could possibly make would be in reference to the female pelvic cavity. In a field where so much work is done at the present day, not only by the gynecologist but the general surgeon, we think a plate showing the relations of the ureter and uterine artery would be greatly appreciated, as the former structure is so frequently injured in doing a hysterectomy. Although the author advocates, as does Kelly, the passing of bougies into the ureters to mark their course, in reality this is seldom done, and frequently it is impossible on account of the tumor which necessitates the hysterectomy, distorting the bladder to such an extent as to render the passage of bougies exceedingly difficult, if not impossible.

The author tells us in his preface that as originally planned the work was to contain 200 illustrations. Vol. III contains 178 alone and the text has been constantly revised and much added, especially with reference to surgical procedure. He certainly deserves the congratulations of the medical profession, particularly the surgeons, for bringing to completion

this work, which has cost him sixteen years of painstaking labor. Every practitioner doing any surgery will find this work of exceedingly great value, and the student of anatomy will find the illustrations a great aid to thorough comprehension of the subject.

CHAS. F. ADAMS.

ORGANIC NERVOUS DISEASES. By M. ALLEN STARR, M.D., PH.D., LL.D. Illustrated with 275 engravings in the text and 26 plates in colors and monochrome. Lea Brothers & Co., New York and Philadelphia, 1903.

The author of this work is so well known, both at home and abroad, and his experience has been so extensive, that we may expect much from him in a book on nervous diseases. The statements made by him, as he himself says, are based in a large degree upon the pathological and clinical material accumulated during the past twenty years. A careful examination of the work shows that he has carried out his intention of utilizing his personal observations without ignoring neurological literature. The descriptions are clear, and the work, while it will be read with interest by the specialist in neurology, will be easily understood by the undergraduate student.

A short chapter on the structure of the nervous system is the introduction to several chapters on diseases of the nerves. These are followed by chapters on organic diseases of the spinal cord and of the brain. The neurone doctrine is taught.

In regard to trophic lesions, Starr says that the explanation of these changes is to be found in the interruption or perversion of sensory impressions and that "nature, lacking its accustomed guide to repair and misled by abnormal impressions, produces effects which are needless under the circumstances, or fails to produce those which ordinarily would be required." Physicians and nurses may read with prickings of conscience that bedsores may be entirely prevented if by extraneous care pressure and irritation are avoided, as in health would occur by change of position and care of the skin. This is not unlike a statement made by Strümpell years ago, and yet there are still physicians who are unwilling to explain bedsores entirely by mechanical causes. Starr has little belief in the origin of trophic disturbances in alteration of special trophic or of vasomotor nerves.

He has never seen a case of true ascending neuritis, although he has seen many cases of painful affection of the extremities above the level of the neuritis that were hysterical in nature. As regards those cases that are not hysterical, Starr's explanation is that "mild irritation long continued produces in a nerve center a hypersensitive state by what is known as a summation of impulses. In this state slight impressions are believed to be intense and local impressions become generalized and widely referred. This state may be induced by neuritis long continued, and has been mistaken for an ascending neuritis." Ascending neuritis has been regarded by other neurologists as an extremely rare affection, and yet some practitioners seem to make the diagnosis in ignorance of this fact.

Starr believes that neuralgia has, in every chronic case, some organic lesion, and that in acute cases some temporary malnutrition of the sensory fibers is present. It is hard to doubt the correctness of this statement, especially if one has made many clinical and pathological studies. Starr accepts this teaching so readily that he does not hesitate to place neuralgia among the organic nervous diseases. Aconitine is the best remedy, in his opinion, for tic douloureux, and he administers it in pill or tablet form, preferring the French pills of Chapoteaux. Each of these contains 1-500 grain, and the remedy must be used with sufficient frequency to produce constitutional effects. He begins with one pill every four hours and decreases the intervals each day one-half hour, until one pill every two

hours is taken or symptoms are present, such as tingling of the tongue and fingers, a sense of general weakness, and feebleness of the pulse. He combines with the aconitine a fiftieth of a grain of strychnine during the first two days, and then 1-100 of a grain when the aconitine is being given every two hours. Sometimes he substitutes caffeine or sparteine for the strychnine. A cardiac stimulant should be used when large doses of aconitine are given, and sudden muscular efforts should be avoided. Other remedies are mentioned. As regards the strychnine treatment in massive doses, as recommended by Dana, Starr says he has seldom seen any benefit result from this treatment, but has seen severe strychnine poisoning ensue. We should like to have further opinion in regard to the aconitine treatment, because most cases of severe *tic douloureux* are treated successfully usually by operation.

In Starr's experience all syphilitic nervous lesions, whether central or peripheral, yield more promptly to the combined use of inunctions of mercury and large doses of iodide of potassium, than when either is employed alone. While he does not deny the possibility of multiple neuritis occurring as a result of syphilis, and even mentions two or three possible cases he regards this cause as very rare. Alcoholic neuritis, in his experience, as in that of Gowers and others, is relatively far more frequent among women than among men. It is important to emphasize the word *relatively*, as alcoholism is so much more common in males.

Starr does not attempt to distinguish sharply between chronic anterior poliomyelitis and progressive muscular atrophy of spinal origin; indeed, the latter is regarded by him as one of the types of the former. The sharp distinction made by some authors between these two forms of muscular atrophy has always seemed to the reviewer somewhat arbitrary. Amyotrophic lateral sclerosis is described as distinct from the progressive spinal muscular atrophy.

The primary lesion of *tabes dorsalis*, according to Starr, lies in the posterior spinal ganglia and in the ganglia of the cranial nerves, and possibly in the ganglia of the sympathetic nervous system. This is a very positive statement, and expresses an opinion that is by no means universal. According to the figures afforded by Starr's cases, about seventy per cent of tabetic patients have had syphilis. He is unwilling to admit that there is "no *tabes* without syphilis." In regard to the much disputed question of antisyphilitic treatment in *tabes*, he advises that this treatment should be employed whenever a history of previous infection is obtained, or whenever its existence is strongly suspected.

No one doubts that chronic myelitis may develop as the result of an acute myelitis, when a certain amount of damage in the spinal cord is produced, but we have the very positive expression of opinion from Starr that myelitis may be from its onset a slow process, and thus chronic from the start.

The views concerning operation in injury of the spinal vertebræ are conservative and wise, if the reviewer may be permitted an expression of his own opinion. There should be some reason to hope for removal of compression before operation is attempted. "If there is evidence, by X-ray examination," says Starr, "of an existing compression of the cord by displaced bone, this should always be removed. If, however, there is no such evidence an operation should be refused." The reviewer has seen a person with fracture of the vertebræ, who had recovered partially in course of time, placed in a much more serious condition by exposure of the spinal cord. Nothing is said regarding operation upon the spinal column in spinal caries. Laminectomy may cause distinct increase in the severity of the symptoms in this disease, as in a case recently seen by the reviewer.

Much might be written concerning the excellent chapters on the diagnosis and localization of cerebral disease, on the cerebral atrophies of childhood, on encephalitis, paresis, abscess and tumor of the brain, etc., but this review can not be more than a brief notice of a thoroughly praise-worthy book, and a feeble attempt to show that the author speaks in no uncertain terms on many disputed subjects, and reveals a wealth of clinical and pathological observation that makes his text-book one of the best on organic nervous diseases in existence.

The temptation to mention the results of Starr's cases of brain tumors is, however, too great to resist, because it is well known that for a long period this has been one of the cerebral disorders to which the author has devoted especial attention. One must read with a feeling akin to sadness and disappointment that but nine per cent of tumors of the brain are open to operation, and that, therefore, in the vast majority of cases we cannot give the patient any hope.

Let us wish a hearty reception to this recent and most excellent text-book on organic nervous diseases. It is elaborately and judiciously illustrated.

SPILLER.

A TEXT-BOOK OF LEGAL MEDICINE AND TOXICOLOGY. Edited by FREDERICK PETERSON, M.D., Chief of Clinic, Nervous Department of the College of Physicians and Surgeons, New York; and WALTER S. HAINES, M.D., Professor of Chemistry, Pharmacy and Toxicology, Rush Medical College, in affiliation with the University of Chicago. Two imperial octavo volumes, illustrated. Philadelphia, New York, London: W. B. Saunders & Company, 1903. Vol. 1.

The scope of this work is outlined by the editors in their preface in the following words: "The object of the present work is to give to the medical and legal professions a fairly comprehensive survey of forensic medicine and toxicology in moderate compass. We believe this has not been done in any very recent work in English. A number of manuals of limited size and scope have been presented on the one hand, and on the other certain systems of legal medicine of almost encyclopedic dimensions. Both find fields of great usefulness; but there is still left a broad ground intermediate between the two which we trust the present work will fill, and it was in this hope that the book has been planned and executed."

The work is made up of a series of comprehensive articles on the different departments of forensic medicine by men of acknowledged ability and wide reputation, and while a detailed review of the work as a whole is hardly possible in a short space, certain of the articles merit more than passing mention.

The Introduction by the editors contains in a short space much sound advice for both expert and attorney which if more generally followed would do much to relieve expert testimony of the discredit with which it is now too often received. "The Technic of Medicolegal Postmortem Examination," by Ludvig Hektoen might well be read by any practitioner of medicine, especially the country practitioner, who may often be placed in a position where he is forced to do a postmortem for the coroner, for upon his knowledge of methods, especially in poison cases, may rest the conviction of the guilty or the acquittal of the innocent.

Articles worthy of special mention are those on "Identity," and "Sudden Death," by James Ewing; "Death and Injuries by Lightning and Electricity," by Smith Ely Jelliffe; "Wounds," by Lewis Balch; "Speech Disorders," by Frank Warren Langdon; while the views expressed in the articles on "The Stigmata of Degeneration," by Frederick Peterson, and "Idiocy, Imbecility, and Feeble-Mindedness," by Frederick Peterson and

Smith Ely Jelliffe, have already become standard through the former author's text-book of "Mental Diseases."

The article on "Insanity," by J. T. Eskridge, is especially valuable because of the short, clean-cut definitions and clear phraseology. A somewhat unfortunate typographical error, however, occurs on page 630 in the use of the word "effects" for "affects" in the following quotation: "'Wahusinn' is a mental disorder in which the delusions and hallucinations are rapidly combined into a complete whole, intimately associated with strong effects. In Verrücktheit the effects are only accidental elements of the clinical history and disappear rapidly."

An important chapter dealing with a subject quite new to works of this character is that on "The Destruction and Attempted Destruction of the Human Body by Fire and Chemicals," by Walter S. Haines. It is an excellent study founded both on experimentation and several notable cases among which the Luetgert case tried in Chicago in 1897 figures largely.

The work of the publishers has been well done, the paper and print are good, the illustrations numerous and some of them of unusual excellence. On the whole we can warmly recommend the work to those desiring light on the subject matter of which it treats, and only trust that the second volume will maintain the high standard of excellence attained by the first.

WM. A. WHITE (Binghamton).

News and Notes.

DR. F. W. LANGDON has accepted the position of visiting consultant to the Cincinnati Sanitarium since the death of Dr. Everts.

PROF. E. KRAEPELIN, of Heidelberg, has been appointed Professor of Psychiatry at Munich.

DR. ORPHEUS EVERTS, long and favorably known to American alienists, died at his home in Cincinnati on June 19, 1903, in his seventy-seventh year, after an illness of some weeks marked by general failure of nutrition.

Dr. Everts served throughout the Civil War as surgeon in the Army of the Potomac; later he was Superintendent of the Indiana Hospital for the Insane, and was one of the experts called by the U. S. Government in the trial of the assassin of Garfield. He was a welcome contributor to the medical press and to general literature. For the past twenty-four years he has been Medical Superintendent of the Cincinnati Sanitarium.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

GIGANTISM AND LEONTIASIS OSSEA, WITH REPORT OF
THE CASE OF THE GIANT WILKINS.

BY PETER BASSOE, M.D.,

RESIDENT ASSISTANT PATHOLOGIST, PRESBYTERIAN HOSPITAL; ASSOCIATE IN
PATHOLOGY, BUSH MEDICAL COLLEGE, CHICAGO.

The clinical features of this case have been studied by a number of prominent men both in this country and Europe in the course of the past ten years. So far as I know, the first description of the case was given by Dana, in 1893, in an article entitled: "On Acromegaly and Giantism with Unilateral Facial Hypertrophy." (13.) He first reports a case of acromegaly in an Indian 6 ft. 7 in. tall, then in introduction to this case remarks:

"The following case seems to be very apposite in this connection, because together with a gigantic general growth, there is a special hypertrophy of part of one extremity. It might be called a case of somatomegaly, a name suggested to me by Dr. Frank P. Foster, with a hemiacromegaly of the head. In plainer language, the individual is a professional giant with an enormous special development of one-half of the face."

Dana's description is as follows: "Lewis Wilkins, aged nineteen, single; occupation, freak; was born in Minnesota. His father was a native of New York; his mother, of Canada of English stock. His parents were healthy people of average size. He has six brothers and sisters, all of ordinary height. He was the second child. He was always large for his age, though not

remarkably so. He grew steadily, however, until by the age of seventeen he was over seven feet. He is now nearly twenty years old, and has grown one and a half inches in the last year. His present height is seven feet four inches, his weight three hundred and twenty-five pounds. His general proportions are for the most part good, but his feet and hands are particularly enormous, and the left side of the face shows a remarkable osseous hypertrophy, involving the frontal bone, upper and lower jaws. The hypertrophy corresponds closely with the distribution of the left trigeminal nerve. It gives his face a curious twisted and asymmetrical look, which is shown imperfectly in the photograph. The first impression is that he has a right hemiatrophy of the face. Closer inspection reveals, however, an enormous thickening of the left upper alveolar processes. The bone bulges out above the teeth as though he had a gumboil. The palatal arch is also greatly enlarged on the left side. The lower jaw is less affected, but is larger and longer on the left side. The teeth are white and even, and are not enlarged. The orbits are alike, but the left brow, and, indeed, the whole frontal bone bulges out so as to give a curious deformity to the skull. The thickening reaches back as far as the coronal suture and stops there. The head in brief is large, measuring 65.5 cm. in its greatest circumference. The osseous hypertrophy makes the naso-bregmatic arc very large, vis., 18.5, while the binauricular arc, measured through the bregma, is relatively smaller, as it does not go over the hypertrophied area. The measurements are: Greatest circumference of head, 65.5 cm. ($25\frac{3}{4}$ in.); naso-occipital arc, 43 cm. (17 in.); naso-bregmatic arc, 18 cm. ($7\frac{1}{8}$ in.); binauricular arc, 37 cm. ($14\frac{1}{2}$ in.); from angle of jaw to symphysis of chin, right side, 13 cm. ($5\frac{1}{8}$ in.), left side 18 cm. ($7\frac{1}{8}$ in.), a difference of 2 inches. The circumference of the chest at the mammary line was $47\frac{1}{2}$ in. and the expansion 3 inches. This shows that he has a thorax of not excessive size proportionately. It is a good deal smaller than that of the Indian giant, whose height was 6 ft. 7 in. and whose chest measured 50 in. The hands were enormous, measuring 26 cm. ($10\frac{1}{4}$ in.) from the tip of the middle finger to the process of the ulna; the circumference of the open hand around the middle of the palm, 27 cm. The feet are relatively still larger. He wears a shoe just a foot and a half long, while the actual total length of each foot is 35.5 cm. (14 in.), and the circumference around the instep is 29.2 cm. ($11\frac{1}{2}$ in.). There is no especial asymmetry of physique except in respect to the face, as described. The left shoulder, however, is a little higher; he is decidedly round-shouldered, and there is a slight dorsolumbar lateral curvature of the spine. He has no cutaneous eruptions, no pigmentation or discoloration. He

has thick, coarse hair, but no beard. His muscular system is but moderately developed; the grasp of his hand is weak; he does not like to climb stairs; he has not much strength. He has good co-ordination; is a good shot. His knee-jerks are slow and feeble. Vision is good in both eyes, and he has no contraction of the visual field. The pupils react normally. The eyes are small, the palpebral fissure measuring 3 cm. His intelligence is good. He sleeps well and eats well. He has a prodigious appetite, and on one occasion ate 27 plates of ice cream at one sitting, thereby winning a wager that he could eat more than two men. No unpleasant after-effects were reported. He has slight headaches at times. His pulse beat and respiration and his heart action were normal. I could not say whether the thyroid was changed in size. It is certainly present." Dr. Dana, in conclusion, remarks: "The interest of this case lies first in the giant growth, and next in the progressive facial hemihypertrophy. That giantism is sometimes associated with acromegaly has been shown by my own case and that of others cited. This patient has some symptoms belonging to acromegaly, viz., the enlargement of the bones of the left side of the face, beginning at about puberty, the kyphosis and scoliosis, the enormous feet, the coarse hair, feeble muscular development, and prodigious appetite. The progressive facial hypertrophy is very interesting on account of its rarity and its association with giantism."

On April 24, 1896, Mr. Wilkins was presented to the Vienna Medical Society by Lamberg. The proceedings as given by the *Wiener klinische Wochenschrift* (29) were as follows:

"Lamberg presented Wilkins. The man, age twenty-two years, has healthy parents. Growth was normal until the fourth year. Then he grew rapidly until the sixteenth year. At eighteen he reached the present height, 245 cm. He is 20 cm. taller than the giant skeleton in the Vienna Anatomical Museum. He presents some skeletal abnormalities, viz., scoliosis, asymmetry of the pelvis, and a tumor of bony hardness, originating from the left upper jaw, which perhaps also causes a narrowing of the left nasal cavity. There is also considerable swelling of the left half of the forehead. The intelligence is proportionate to his age. The genital functions are normal. Lamberg considered the case as one of acromegaly.

"M. Sternberg: Only three skeletons in museums are larger, the Irish giant at Dublin (259 cm.), one at St. Petersburg (254 cm.), Patrick O'Byrne, London (249 cm.). Giants frequently present morbid features, general dystrophies or vegetative disturbances. About 40 per cent have acromegaly. Doubtless there are

also quite normal giants. He does not consider Wilkins a case of acromegaly. The prominence of the lower jaw is lacking as well as deformities of the soft parts of the mouth and nose; the tongue is normal, the hands are proportionate, etc. Abnormal is the great development of the left half of the face. The lips are also somewhat thickened on the left side, so we may speak of a hemihypertrophia facialis, principally ossea. The deviation of the axis of the left eyeball and the narrowing of the left nasal cavity point to bony changes in the orbit, and the slowness of movements and phlegmatic temperament to intracranial hyperostoses and exostoses. Buhl's case, Hassler, is probably the most analogous of the known cases.

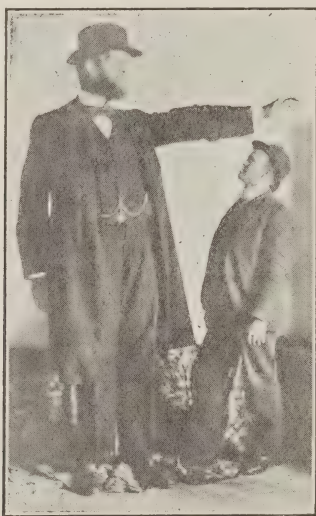


Fig. 1 Mr. Wilkins and his brother.

"Schlesinger accentuated that acromegaly was clinically sufficiently sharply limited to exclude this case. Above all, the enlargement of the soft parts, almost always present in acromegaly, was lacking. The lips and tongue are approximately normal. So is the position of the teeth, which is almost always altered in acromegaly on account of the enlargement of the lower jaw. Schlesinger asked about the eye findings, as ocular disturbances are frequent in acromegaly, such as bitemporal hemianopsia, optic atrophy and paralysis, due to the frequency of hypophysis tumors.

"Lamberg: 'The eye findings are normal.'"

In his exhaustive treatise in Nothnagel's "Spec. Pathologie,"

Sternberg (43) again discusses this case and groups it together with Buhl's and one of Sirena's as "pathological," non-acromegalic gigantism, the "pathological" features in this group being tumor-like exostoses (leontiasis ossea and hyperostoses). In connection with leontiasis he says (p. 86): "If the disease is extensive and associated with gigantism, a confusion with acromegaly is likely, as actually happened in the case of the giant Wilkins (Lamberg). Here was to be considered that the enormous hands and feet were proportionate to the excessive size of the body, and that a close examination showed the presence of bone tumor, with absence of acromegalic changes of bones and soft parts."

Schmidt, in his treatise on gigantism (41) takes the same view of the case, being positive that this case is not one of acromegaly, but analogous to that of Buhl. The latter case will be considered in detail later.

Mr. Wilkins was admitted to the service of Professor Bevan at the Presbyterian Hospital, June 28, 1902. The writer wishes to thank Professor Bevan for his kindness in allowing him to use the following history, which was obtained by the interne, Dr. Pearsall:

Lewis Wilkins, age 28. Family history: Father, age 55; mother, age 60; both well. Four brothers and one sister alive and well. All relatives and ancestors, so far as known, have been of normal stature. No history of tuberculosis or tumors in the family.

Personal—Patient weighed 9 pounds at birth, and was normal in size until four years of age, when he began to grow rapidly, and at ten years was over 6 ft. in height, and continued to grow until about 18, when he attained his present height (8 ft. 2 in.). He has been on exhibition the past twelve years, and has traveled extensively in the United States and Europe.*

Usual diseases of childhood. Malaria one year ago. Lateral curvature of spine for about twelve years. When eight years old he noticed a growth, size of a small marble, above the left eye. This has continued to grow slowly until four or five years ago, since which time he thinks it remained stationary until three months ago, from which time it has increased in size rapidly. About two years ago, he states, he saw Professor Virchow in Berlin, who, so he says, pronounced the growth a benign one, said it was on the external surface of the skull

*In a pamphlet distributed at Mr. Wilkins' exhibits in Germany it is stated that he did not grow in height from the age of 18 to 21, but gained 3 inches from 21 to 22, when he reached 245 cm. It is also stated that his father measured 161 cm., his mother 170 cm. in height.

and would probably never cause him trouble. Gonorrhea four years ago, discharge lasting about three weeks; denies other venereal infection. Three months ago he would have severe pain in left frontal and temporal region each morning on rising, pain disappearing gradually by 10-11 o'clock. For the past month he has had continued pain in this region when awake and has required the constant use of opiates. About one month ago he lost the vision in the left eye. Thinks it has been weak for several years. One week ago he also lost the sight in the right eye. Says vision disappeared suddenly; saw dimly on retiring one evening and the next morning could not appreciate light. He has had several spells of vomiting during this time. These spells have not been closely connected with eating. He states that he has had no sensation over the left side of the face for about one month, and during this time has had difficulty in speech, his voice being thick and heavy. Two months ago he experienced ringing sensations in the left ear and has since complained of earache. Says he is deaf in the left ear. Since his arrival at the hospital he has been dull; almost semi-stuporous. Has had several involuntary bowel movements. He was taken with diarrhea while on the way to Chicago from his home in Oklahoma.

Physical Examination. Height 8 ft 2 in., according to statement of patient. Measures 56 in. around the chest. He is well proportioned. There is a growth on the left side of the head which extends from the median line outward and backward as far as the external auditory meatus and downward over the face to the alveolar process of the superior maxilla. This mass is firm except in an area a little larger than a silver dollar in front of and above the external auditory meatus which is soft and semi-fluctuating. The left eye is closed. The left upper lip is thicker than the right and droops. The pupils react neither to light nor distance, and the left eyeball is immobile. The tongue does not deviate on protrusion, and the muscles on both sides act in frowning or laughing. There is an area of anesthesia extending from the left ramus of the lower jaw to the external auditory meatus, and upward to the median line of the skull. The left half of the tongue is also devoid of sensation except for a narrow strip near the median line. 6-28, urine 1027. Acid. No albumin or sugar. No casts. Red cells and leucocytes present. 6-28, blood examination, hemoglobin 85 per cent; reds, 4,876,000; leucocytes, 9,300. Temp., 98.4-101.4; pulse, 80-92 on day of admission. 7-1, urine, 2,400; cc., 1,010, ac. No albumin or sugar. Urea, 1.75 per cent; temp., 98-100. Ophthalmoscopic examination showed a bilateral choked disc.

The diarrhea became more severe while in the hospital, the

stools were slightly bloody, pain and tenesmus were present. He gradually failed, and died July 10, 1902.

The autopsy was held immediately after death by Professor Hektoen, assisted by Dr. L. Loeb and myself. A relative who was present did not permit any measurements to be taken. Fortunately those taken by Dana nine years ago give us the relative proportions. A growth in height has undoubtedly taken place since that time, as Lamberg's measurements and the patient's statements show; also an increase in the circumference of the chest. The time allowed for the autopsy did not permit a more detailed description of the external appearance of the body than that given in the clinical notes.

After the removal of the thoracic and abdominal viscera both carotids were injected with 4 per cent formaldehyde solution in order to harden the brain *in situ*.

Anatomic Diagnosis.—Necrotic and ulcerative colitis and ileitis; cirrhosis of liver; chronic catarrhal gastritis; epitheliosis of esophagus; hemorrhagic broncho-pneumonia (right lower lobe); enlargement of thyroid; sarcoma in region of hypophysis; extension of tumor to subcutaneous tissues; diffuse hyperostosis of frontal, left parietal, left temporal, and left superior maxillary bones; calcification of left pleura and spinal arachnoid; compression of brain; general gigantism.

The abdominal cavity is empty and the peritoneum smooth and free from adhesions. The diaphragm reaches to the fourth interspace on both sides.

Both pleural cavities are empty and there are no pleural adhesions, except between the right upper and middle lobes.

The pericardium is smooth and the cavity contains no fluid.

The thyroid weighs 112 grams; it is uniformly enlarged, and glistening on the cut surface as if the follicles were dilated with an unusual amount of colloid material. In the left lobe are two small, circumscribed, somewhat reddish nodules.

There is no trace of the thymus in the corresponding fatty masses.

The larynx and trachea show no changes.

The lungs are crepitant throughout, much lighter and spongier than usual, especially in the case of the left lung. In the lower right lobe are two hemorrhagic areas with elevated bases, the longest diameter being 6 cm. In the pleura over the lower left lobe is a flattened calcareous district, 1 cm. square, and at the right of the left lung are calcareous glands.

The heart is normal in shape and weighs 465 grams. There are no changes in the endocardium. The myocardium is firm and reddish brown.

The aorta is quite smooth throughout its entire extent.

The spleen is large and its capsule smooth. Cut surface is reddish and of normal appearance. The spleen weighs 620 grams.

The lymph nodes in various parts of the body are not enlarged.

The pharynx is normal.

The esophagus shows a number of flattened, whitish, firm, raised, nodular areas in the upper part of its course, generally arranged somewhat linearly. Otherwise the membrane is smooth.

The stomach is very large. It weighs, including the duodenum, 655 grams. The mucous membrane is thrown into huge wrinkles, not unlike cerebral convolutions, which cannot be straightened out on stretching the muscular coats. The mucous surface is covered with a turbid, tenacious mucus.

The small intestine from the duodeno-jejunal junction to the ileo-cecal valve, measures 20 meters in length. The mucosa is normal in appearance until within about 50 cm. of the valve, where it appears granular, rough, and of a dirty greenish yellow color in areas, with rather superficial small ulcers. Just above the ileo-cecal valve the entire circumference of the ileum, for about 5 cm., is intensely necrotic.

The large intestine is very voluminous; it measures in length, approximately, 4 meters. Its entire mucous membrane from the ileo-cecal valve to the anus is the seat of necrosis in the form of grayish, dirty, granular points and areas, and more especially of thickly disseminated, rather small ulcers with smooth floors as a general rule, and overhanging margins. In many cases the undermining of the margins is so extensive that large submucous tunnels are formed and neighboring ulcers may communicate with one another by means of these passages. In the flexures, and especially in the rectum, congestion of the mucous membrane is noticed.

The liver is large, weighing approximately 4,000 grams; it has a granular and nodular surface, especially near the lower margin of the right lobe. There is some increase in consistency and more or less distinct breaking up of the surface into large islands of liver tissue by fibrous bands. The under surface of the liver shows anomalous formation of the lobes, the right being taken up largely by a large projecting mass with some constriction at the base of its junction with the main part of the liver.

The gall bladder and bile passages do not show any changes.

The pancreas is large, weighing 275 grams; it is normal on the cut surface.

The ardenals are large, weighing 42.5 grams; they do not show any changes on the cut surface.

The kidneys also are large, weighing together 525 grams.

Capsule is free except over a rather large area in the left kidney, where there is an irregular, radiating, depressed scar. The surface is, in general, smooth. The cortex is unusually wide; the cortical markings distinct, and the glomeruli visible as reddish points. In the apices of several pyramids are small deposits of a granular, sand-like material.

The mucous membrane of the ureters and of the bladder is smooth. The bladder is distended with urine and is very large.

The prostate is not as large, in proportion, as other organs. The same seems to be true of the seminal vesicles and testicles, the latter weighing 85 grams.

There is a soft subcutaneous nodule almost as large as a fist

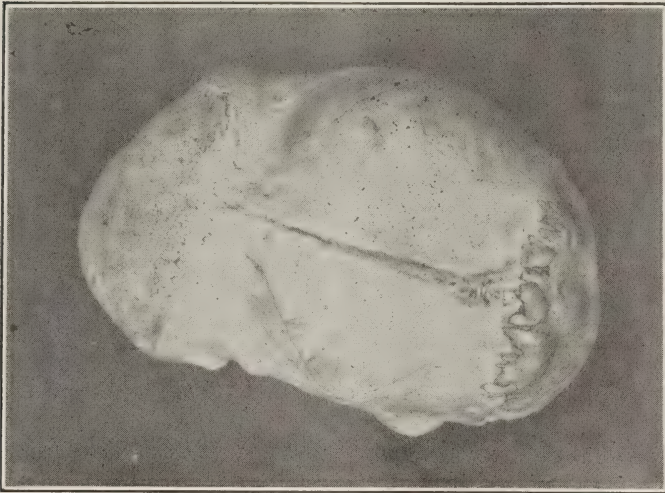


Fig. 2 Outer surface of skull-cap.

behind the left ear; and another of greater firmness involving about two thirds of the left side of the forehead. The tumor tissue on section is pink and homogeneous. The frontal region of the skull is very thick, especially on the left side. The skull cap weighs 1,450 grams; it is 68 cm. in circumference, 25 cm. in length; from the obliterated interfrontal suture, lower end, to the right coronal suture, internal horizontal measurement, is 5.7 cm.; from the same point to the left coronal suture is 8.5 cm. In the median line the frontal bone is 8.5 cm. thick, and is softer than normal, especially on the left side. There is marked asymmetry, the interparietal suture being displaced to the right. Fissures for vessels are relatively shallow. In front of the coronal suture the

bone is rough from the presence of thickly-set depressions, varying from 0.5 to 3.0 mm. in diameter. The remainder of the skull cap is smooth. The thickness varies from 2 to 8 mm. On the outside the interparietal suture presents a prominent ridge in its posterior half (see photograph). The parieto-occipital suture is also prominent. The base of the skull is greatly deformed. On the left side the anterior fossa is obliterated by the thickened frontal bone, and the middle fossa also is almost filled in with bone.

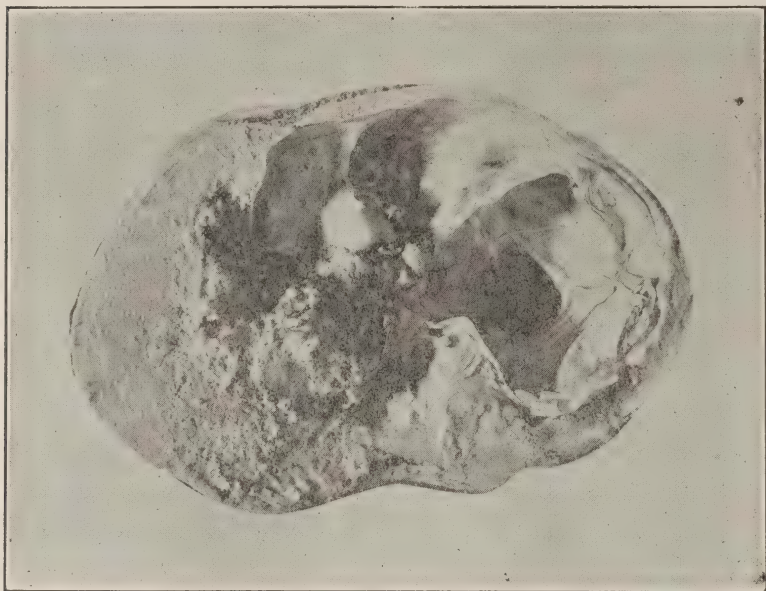


Fig. 3 Base of skull.

The sella turcica is wide, its floor partly eroded. In this region is a large tumor mass which has grown into the pharynx, orbits, and ethmoidal sinuses, and has destroyed the roof of the nose; the roof of the left orbit has been much more extensively invaded than the right. In the median line of the surface of the tumor exposed at the base of the skull after removing the brain is a projecting pedicle, darker than the tumor mass. This is found to be the infundibulum. It measures 3 mm. in diameter and is inserted into a round elevation 13x6 mm. of the same color as the stalk. The tumor itself is white on the surface, lobulated, the lobules 2 to 4 mm. wide, separated by narrow, but deep, fissures. On section the superficial part of the tumor is white and distinctly lobular. A gelatinous fluid exudes from the cut surface. Deeper down it is

more homogeneous and there is more slimy fluid. Still deeper dark red bands permeate it. The portion of the tumor removed with the hypophysis weighs 150 grams, but at least as much more was scooped out in pieces or left behind.

The brain weighs 1,540 grams. The distance from the frontal to the occipital pole is 18 cm.; transverse diameter, 14.5 cm. There is marked deformity of the left hemisphere to correspond with the obliteration of the anterior fossa of the skull. The left frontal lobe has been pressed upward and to the right. It termi-

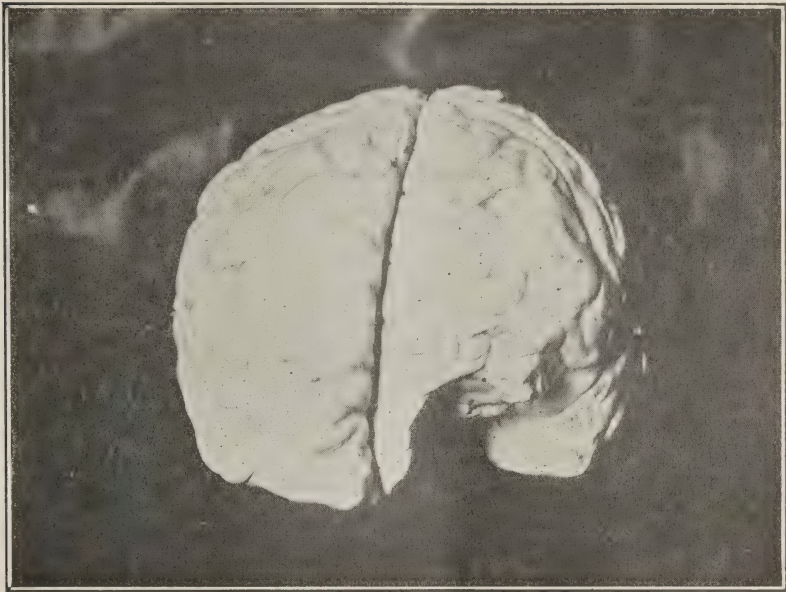


Fig. 4 Anterior view of brain.

nates in a narrow strip along the median fissure. The lower end of the strip is 8 mm. above the pole of the right frontal lobe, its width 15 mm. at the base, 8 mm. at the lower end (see photograph). Farther to the left the brain substance has been compressed so that the inferior surface of the left frontal lobe is 3.5 cm. higher than that of the right side. The left temporo-sphenoidal lobe also is smaller and compressed; its inferior surface measures 1.5 cm. antero-posteriorly, while the right measures 3.7 cm. The convolutions of the brain are generally broad and flattened; the sulci shallow (see photograph). The left optic nerve is narrower than the right. The other cranial nerves appear symmetrical.

The length of the medulla and cord, from the pons to the filum terminale, is 60 cm. Below the mid-thoracic region are many thickened, partly calcified patches on the inside of the dura. These are rough and granular on the inner surface; the attached surface, on separation from the dura, is smooth. The largest of them measures 3x1.2 cm. To one of them a nerve-root is adherent.

The sternum is 26.5 cm. long, the greatest width of the manubrium 10.5 cm., of the body, 5.5 cm.

Histological Examination. The soft tumor. All parts of it



Fig. 5. Magnified 8 diameters. Showing relation of hypophysis to surrounding tissues, mostly tumor.

were subjected to examination, and its structure was found to be essentially the same everywhere. It is made up of cells, mostly spindle-shaped, with large oval or round nuclei. Karyokinetic figures are numerous. The protoplasm of the cells stains faintly. There is no suggestion of glandular or other epithelial structures, the cells having no definite arrangement. The texture is loose, there being a large amount of finely granular intercellular substance, stained lightly by hematoxylin. This is evidently in part the coagulated product of the abundant viscid material noted on gross examination. In the deeper part of the tumor the degenerative changes are more marked. There are large necrotic areas,

and also hemorrhages. The reason for the lobulated appearance of the surface is found in the presence of fibrous bands running perpendicularly to the surface. Such trabeculæ are also seen in the deep portions. In places tumor cells, sometimes with karyokinetic figures, lie in them. In various parts, more particularly near the hypophysis, are bony spicules, some of which are rich in bone corpuscles, and probably newly formed. Here and there giant cells are seen. The tumor may be designated an osteoplastic sarcoma with edema and mucoid degeneration.

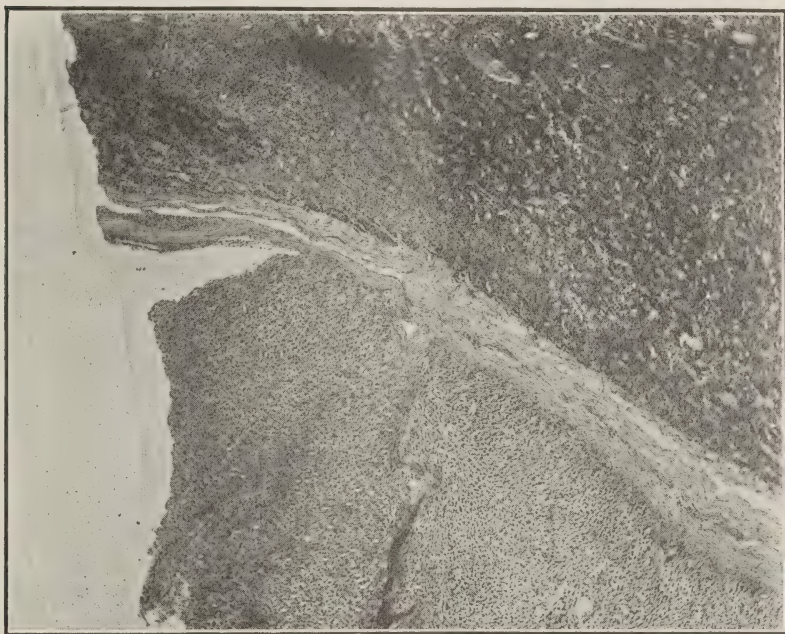


Fig. 6. Magnified 45 diameters. On one side of dura hypophysis tissue. On other side tumor with bony spicule.

The Hypophysis and its Relation to the Tumor. Serial sagittal sections* were made of the lateral halves after a preliminary bisection through the infundibulum. A fibrous membrane (dura mater) was found separating the hypophysis from the tumor, the sharp border being plainly visible to the naked eye on account of the difference in staining. At one point on the left side the tumor and the hypophysis are in direct contact, but the border is sharp, and there are no transitional cell forms. Some enlargement of the

*The writer wishes to thank Messrs. Foreman, Jackol, and Darmer for valuable aid in this tedious work.

organ is present, the greatest length as seen in the sections being 20 mm. At this point it measures 8 mm. from above downwards. It appears flattened in all the sections. The histologic structure is essentially normal. There are the usual large, highly eosinophilic cells, arranged in elongated columns, or in clusters of about the size of renal glomeruli, and smaller cells with cytoplasm stained a faint blue with hematoxylin, and rounded nuclei, also with more or less glandular arrangement. In the extreme right portion of the anterior lobe this is the only cell type seen, and glandular ar-

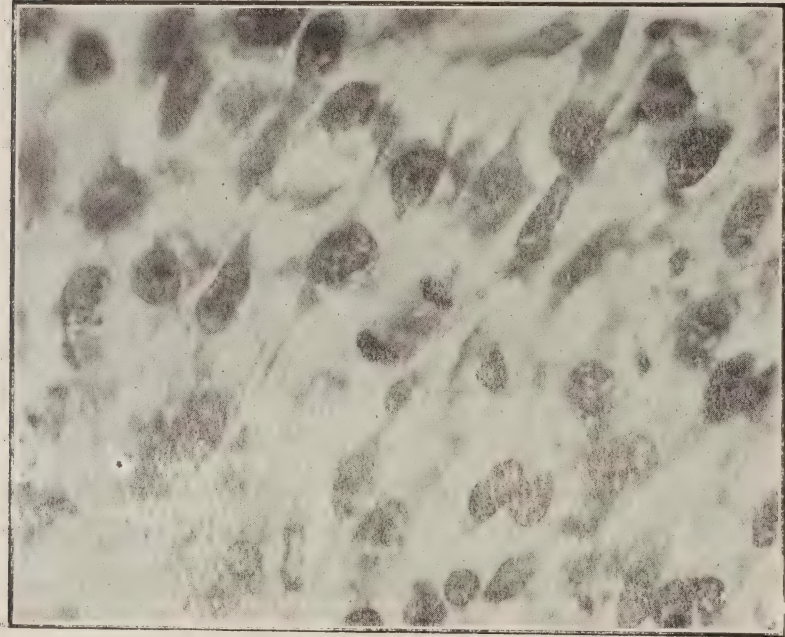


Fig. 7. Magnified 1,200 diameters. Showing finer structure of tumors. One mitotic figure in the center.

rangement less apparent. There is a moderate number of spaces containing a colloid material and lined by epithelium. Engorged vessels are abundant. There are also numerous hemorrhages, a few foci consisting of densely-packed, small, round, deeply-stained mononuclear cells, an occasional amyloid body, and in places the large eosinophilic, epithelial cells are vacuolated. In places the trabeculae and adventitia of the small vessels have a hyaline appearance. There are generally deposits of pigment at the margin of such areas, indicating that they probably mark the site of old

hemorrhages. The posterior lobe is small and made up of a faintly fibrillar ground substance, stained lightly by hematoxylin, and containing a moderate number of long and narrow, deeply-stained nuclei; small vessels are numerous and engorged.

The Skull. Section from frontal bone in median line: Less than half the section is compact bone in irregular islands, the remainder a loose reticular tissue, consisting of a fibrillar unstained matrix with a small number of long, narrow, nucleated cells. At one edge of the specimen the interstices between the islands of

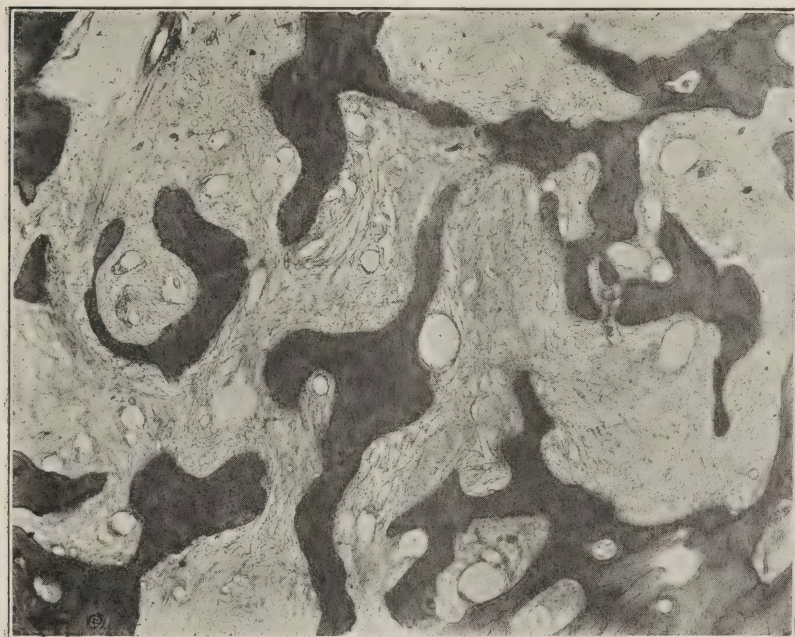


Fig. 8. Magnified 38 diameters. Frontal bone in median line.

bone are densely packed with spindle-cells like those in the soft tumor.

Left side of frontal bone near external surface: Here the compact bone is even smaller in amount, lamellæ imperfect. Haversian canals large and numerous. Most of the bone appears to be of recent formation judging from the number and size of the cells. Abundant osteoblasts are seen, mainly consisting of spindle-cells, with frequent mitotic figures. The texture of the tumor tissue in places is very loose, suggesting mucoid degeneration or edema.

Section from inner surface of frontal bone differs from the

preceding by the larger amount of deposits of new-formed bone, rich in cells, and taking a deeper hematoxylin stain than the old bone. They form small round or irregularly shaped bodies, lying together with spindle-cells, osteoblasts, and certain large, faintly-stained oval cells in the spaces bounded by trabeculæ of old bone. In notches in the surface of the latter osteoclasts are occasionally seen. Right sphenoid bone. Here the bony trabeculæ are also scarce, and enclose tumor tissue everywhere. In places are islands



Fig. 9. Left side of frontal bone, showing sarcomatous invasion.

of new-formed bone surrounded by osteoblasts and a few osteoclasts. Sections from a rib show nothing abnormal.

The ganglion cells of the cerebral cortex are practically normal. A few of them contain yellow pigment with lateral displacement of the nucleus. Marchi preparations of the cortex and subcortical white matter show no degeneration. There is a moderate amount of diffuse degeneration in the lower medulla and cervical cord. The roots are also slightly affected. In the cervical spinal ganglia a number of amyloid bodies are seen, the vessels of the capsule have thickened hyaline walls, a few of the small vessels contain thrombi. Many of the ganglion cells contain large masses of yellow pigment. The nuclei are distinct, generally centrally

located; their staining is very variable in intensity. There is some thickening of the smaller arteries in the lumbar region of the cord. The calcified patches in the spinal meninges are found to contain true bone. Both optic nerves are extensively degenerated, more so the left, as seen in Marchi preparations.

Thyroid. The acini are generally distended with homogeneous colloid material. In places several acini appear to have coalesced. The blood vessels are few and small.

Lung. In a section from the lower lobe of one lung the alveoli are completely obliterated, so that it is difficult to recognize lung structure. In most places the lung tissue is replaced by round and elongated connective tissue cells. Here and there are considerable areas of blood which is partly in blood vessels and some apparently free in the tissues. The connective tissue is well organized into strands in places; in other places the organization has not gone so far. In one small area the alveolar walls may be made out, and the centers of the alveoli are filled with red blood corpuscles and leucocytes. Developing capillaries are seen in the tissue. These are filled with corpuscles. There are some pigment patches near the blood vessels. The pleura is thickened and greatly congested.

Heart. In sections of the heart muscle there is seen a very slight increase in interstitial tissue; otherwise there are no changes.

Spleen. The spleen is much congested, red corpuscles being diffused quite uniformly throughout the tissue. Malpighian corpuscles are clearly marked.

Lower Ileum. In sections examined, the mucosa is absent. The submucosa is greatly infiltrated with round cells. Blood vessels are congested, and in some places there is hemorrhage into the tissues. In places the submucosa has undergone necrotic change and sloughed away down nearly to the muscular layer. The circular layer of muscle fibers also is infiltrated with round cells and in many places there are blood vessels distended with blood. In the submucosa can be seen thrombi, consisting of layers of fibrin, between which are red and white corpuscles. Fibrin also is deposited in shreds throughout the submucosa.

Colon. The mucosa is much eroded in most places, and in some entirely absent. The surface is necrotic, so that only the lower parts of the glands are seen. The lumina of many of the glands contain mucous material. There is considerable round-cell infiltration. There is also hemorrhage into the tissues, and the vessels are greatly engorged. This condition extends also in the submucosa as far down as the muscular layer. Throughout the submucosa there are shreds of fibrin arranged usually in an ir-

regular network. Near the mucosa an amoeba may be seen occasionally.

Rectum. The rectum presents much the same appearance as the colon, except that the condition is much more marked. The mucosa has undergone necrosis, and sloughing has occurred. There are large spaces near the surface, some of them filled with blood and fibrin; others are empty. In some places the blood is confined to blood vessels, but frequently it is diffuse. There are numerous round cells, some polymorphonuclear leucocytes, and plasma cells. Amoebæ are present in large numbers, most abundant in the upper part of the submucosa, but are also found in the other coats. They are usually spherical, but occasionally oval or oblong. They stain with eosin faintly. Usually the nucleus can not be seen, but frequently it is quite distinct, though not stained with hematoxylin. The amoebæ are invariably vacuolated. They are found mostly in the solid tissues, but occasionally may be seen in the blood vessels or large lymph spaces.

Liver. Interlobular connective tissue is increased in amount and distinctively isolates the liver lobules; this connective tissue does not extend into the lobules. The bile capillaries and blood vessels in this tissue show no changes. At the margins of the lobules the liver cells are not as distinct as elsewhere, and seem somewhat compressed. Elsewhere the liver cells are normal and the nuclei distinct. Occasionally fat vacuoles may be seen. The capsule of the liver is considerably thickened.

Pancreas. The pancreas shows no changes. Numerous areas of Langerhans are present.

Adrenals. There are a few areas of round-cell infiltration in the medulla and in the zona reticularis. Under the capsule in the cortex, there is seen congestion of the blood vessels.

Kidney. The glomeruli completely fill their capsules in most cases. Many of them are congested and show round-cell infiltration. The capsule is somewhat thickened. In places the capillaries between the tubules are much congested; there are also many round cells in places. The renal cells are large, cell walls distinct, and nuclei clear and distinct. In the tubules is seen a small amount of granular material. In some places the cells lining the tubules are slightly swollen and granular. Frequently clear spaces are seen around the nuclei. In other tubules the lining cells are low and flat, the nuclei stain deeply, and the lumen of the tubule is filled by a granular material.

Testicle. The seminiferous tubules show quite marked changes. Just inside the basement membrane the sustentacular and spermatogenic cells have undergone a hyaline change. In some tubules the hyaline change has advanced to the center, while in others the

hyaline area separates the normal cells from the basement membrane. Some tubules are apparently normal. There is some increase of fibrous tissue, and a few areas of round-cell infiltration.

A *bacteriological examination* was made by Mr. E. H. Ruediger of Rush Medical College, who kindly reported that no growth was obtained from the heart's blood, and that two organisms were isolated from the intestinal ulcers, one a typical colon bacillus, the other an unidentified bacillus, which possessed some characteristics both of the Shiga bacillus and the ordinary colon bacillus.

A *chemical examination* of the thyroid gland was made by Dr. H. G. Wells, who kindly furnished the following report:

"One-half of the gland was received in alcohol, with the statement that the gland was symmetrical. The tissue was ground up finely in a meat chopper, and to the ground material was added the alcohol in which the tissue had been preserved; the whole was then evaporated to dryness on the water bath, and then dried to a constant weight over sulphuric acid. The total weight of the dried gland so obtained was 13.80 grams, giving a total for the entire organ of approximately 27.60 grams. The normal weight of dried thyroid glands of residents of the United States has been found to be in the vicinity of 5 grams.* Portions of the dry powder were then analyzed by the modification of Baumann's method, and the average amount of iodine of four determinations found to be 2.28 mg. per gram of dried substance. The total amount of iodine in the entire gland was, therefore, 62.928 mg. Analysis of the glands of a series obtained in Chicago showed the average amount under normal conditions to vary from 1.5 to 2.5 mgs., with a total iodine content of about 10 mgs. Similar results were found with glands removed from residents of other parts of the United States. It is seen, therefore, that the gland of the giant contained a normal amount of iodine in each gram of gland substance, but on account of its greatly increased size the total amount was six times as much as normal.

"Histological examination of the gland explains fully the analytic results. It is seen that the enlargement is of the type of simple colloid goiter, characterized by large aveoli distended by a normal colloid material. It has been shown by Wells (*loc. cit.*) and corroborated by A. Oswald,** that in goiters as a rule the amount of iodine is in direct proportion to the amount of colloid. In the goiters of the "parenchymatous" type characterized by a great hyper-

*"The Physiology and Therapeutics of the Thyroid Gland and Its Con-
geners." H. Gideon Wells, Journ. Amer. Med. Assoc., Act. 30, Nov. 6 and
13, 1897.

**A. Oswald, Virchow's Archiv., 1902, 169, p. 444.

plasia of epithelial cells, and containing but little colloid, there is a marked decrease in the amount of iodine in each gram of gland, corresponding to the small amount of colloid, but the total amount of iodine is about normal because of the great size of the thyroid. On the contrary the glands of which this is a type show a colloid containing about normal quantities of iodine in each gram, and consequently with a greatly increased total amount. When the amount of colloid is very large Ostwald has found that part of it may not contain iodine. It seems possible that the parenchymatous goiters may be the result of a deficiency in supply of iodine in a condition to be transformed into thyroiodine, so that the gland hypertrophies in the effort, which is apparently successful, to obtain enough iodine for the requirements of the body. The colloid goiters may result from an increased requirement for thyroiodine in the economy, with normal supply of iodine, leading to the formation of much colloid with a normal proportion of iodine so long as the supply is adequate. That this giant, particularly with a diseased hypophysis, should have a large amount of iodine in his thyroid was to be expected and agrees with the theory. The result also shows that in this case of giantism the goiter was not different from simple goiter, at least so far as histology and iodine content indicate."

(To be continued.)

STUDIES UPON THE CEREBRAL CORTEX IN THE NORMAL HUMAN BRAIN AND IN DEMENTIA PARALYTICA.¹

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Introductory. The following contribution to the study of the cerebral cortex in the normal human brain as compared to that in dementia paralytica, does not include much that is new to the pathology of the latter, but aims to present the subject in a somewhat different manner than has heretofore been done. The writer's perusal of many anatomical and pathological articles has often been unsatisfactory to himself, owing to many generalized statements of conditions found without any adequate illustrations of what was really to be seen. It has been the endeavor in this article to present and describe photomicrographs of actual sections from a practically normal human brain histologically by way of comparison, and to follow these by photomicrographs of sections of a brain of a well marked case of dementia paralytica. These photomicrographs have been supplemented by drawings. The possibilities as well as the limitations of photomicrography in preparations stained by the Nissl method, it seems to the writer, are fairly well brought out in this article, and this was one of the objects sought in its presentation. That photomicrographs show structure and arrangement in but one plane of limited thickness is true. It is also true, however, that they show exactly what is there and the exact relation to other parts, and there is no personal equation such as is bound to occur in any drawing. A drawing, on the other hand, is always more or less composite, does not show exact relationship, and involuntarily the point to be illustrated is made to assume undue prominence. In work of this kind artefacts are constantly to be looked for and avoided, and the entire process of preparation, as well as the history of the case, must be also carefully considered before any conclusions should be drawn.

¹Submitted in partial fulfilment of the requirements for the degree of Doctor of Philosophy in the Faculty of Pure Science, Columbia University.

When it is considered how extremely thin the human cortex is, 1.5 to 3.5 millimeters; that its exterior surface is estimated at about 200,000 square millimeters; that it is intricately arranged in convolutions of varying size and direction; that it is exposed to injury prior to death, at the autopsy, during transportation, and also in the various manipulations of fixation, imbedding, sectioning, staining, decolorizing and mounting, it can readily be seen how artefacts may result by the time the specimen is prepared for examination. This was forcibly brought to the notice of the writer in the examination of some sections but a short time ago. Owing to a dull microtome knife the nuclei of a number of the cells were completely carried outside of the cell-body and the cell-bodies themselves broken and cracked in places. This was at first regarded as a pathological process until attention was called to the fact of the nuclei all being extruded in the same direction, and the breaks and cracks presenting a ragged and irregular appearance. Even when the greatest care is used there will be a variation in thickness in different sections—one being somewhat thinner and the next correspondingly thicker than actually registered by the microtome, although most carefully imbedded, so that variations in the thickness are also to be considered. Furthermore, in fixation, shrinkage or swelling may occur, the latter being demonstrated by Donaldson (1891), who placed fresh nerves in a solution made up of 2.5 per cent of potassium bichromate, plus one-sixth its volume of 95 per cent alcohol for three weeks. He then washed them for one day in water and placed them in 95 per cent. alcohol for three or four days, and finally in 80 per cent alcohol, using six olfactory bulbs, six olfactory tracts, and three pairs of optic nerves of the sheep. This was exactly the same treatment to which his specimens of human nerves had been subjected. The volume of specimens of olfactory bulbs subjected to this treatment and afterwards placed in 80 per cent alcohol was found to be 5.2 per cent greater than fresh specimens, those of the olfactory tracts 8.8 per cent greater, while the optic nerves showed but 2.6 per cent increase in volume. Imbedding in celloidin did not affect the volume, while some stains, as acid fuchsin, produced no change, whereas Delafield's hematoxylin caused swelling, and thus

increase in volume. Hammarburg (195) found by experimenting with small blocks of cortical tissue that by accurate measurements blocks hardened in Müller's fluid for three days showed a volumetric increase of 11.7 per cent. Other blocks hardened in Müller's fluid for three weeks, and then, transferred to alcohol and left in the latter for fourteen days, showed, on the other hand, a shrinkage in volume of 43 per cent. In alcohol, 95 per cent, at the end of twenty-four hours, a shrinkage in volume of 20.5 per cent was observed. This shrinkage is furthermore found to vary with different blocks from the same region of the same brain, and it is found that there is no constant relation between pieces from brains of various grades of hardening. Thus the method of fixation must always be taken into consideration. Again, in staining under exactly similar conditions, one section on a slide will be found more intensely stained than another section on the same slide, or even one part of a section will be found more intensely stained than some other part of the same section.

In the subsequent decolorization still greater variations may occur, for, if carried too far, important structures may be completely decolorized. If carried to the other extreme it is impossible to differentiate the various parts. The only way to avoid either extreme is by repeated examination of the sections under the microscope while the process is being carried on, as under similar circumstances one section in a given time may become much more decolorized than another. In regard to mounting of sections stained by the Nissl method, as all were that have been employed in this article, xylol-damar has given the greatest satisfaction to the writer. With this mounting medium there was no subsequent shrinkage, and thus a resulting exposure of the sections, also no decolorization was to be observed at later periods. In some sections mounted for over three years, no decolorization was observed, even after several exposures to the intense light necessary for photomicrographic purposes, and frequent exposures to light in their examination under the microscope. Canada balsam was also used, but in time the sections were found partially or completely decolorized. Benzene-colophonium has a

tendency to shrink and crack and thus expose portions of the sections in some cases.

It is desired to emphasize the importance of careful attention to all these details at the beginning of the article, in order to impress upon the reader the necessity of the greatest care and conservatism in the interpretation of any changes found in the specimens under observation, and, furthermore, the *correct* interpretation of the same. Before going on to the main subject of the article, a brief historical review of work in this line will be given.

Historical. The first study of the internal structure of the nerve cells began over fifty years ago when Remak (1841) called attention to a fibrillary structure inside the axis cylinder and cell-body of certain nerve cells. Later, Max Schultz (1872) studied nerve fibers and cells from various parts of the central nervous system of different animals with elaborate descriptions of his findings. He dealt with a description of the fibrillary appearance of the interior of the cell-body largely. This doctrine of the nerve cell was afterwards strongly supported by Boll (1873), Schwalbe (1885), and Ranvier (1878). Opposite views were, however, held by Arndt (1874), and Key and Retzius (1876). Arndt, in 1874, described the structure of the spinal ganglion cells and spoke of the presence in them of different kinds of elementary spherules which varied in size and general appearance. Key and Retzius stated that the ground substance of the spinal ganglion cells was homogeneous, but that in it numerous strongly refractive, round, oval granules were present, and they thought the appearance of a concentric striation or fibrillation could be simulated through the arrangement of the granules in rows. Flemming (1882) saw granules within the cells which would stain with nuclear and azo dyes and hematoxylin, but nevertheless affirmed a fibrillary structure of the central cells and of a tortuous or much curved threadwork within the spinal ganglion cells between the granules. In recently published articles (1896) he still maintains that fibrils exist inside the nerve cell protoplasm.

Dogiel (1893) has also expressed himself in favor of the views of a fibrillary structure for certain at least of the nerve

cells. In 1885 Nissl published the first of a series of articles in which he calls particular attention, in tissues hardened in alcohol and stained in basic aniline such as majenta red and methylene blue, to certain structures. By Nissl's method these are brought especially well into view, and their arrangement in the protoplasm and their significance for the function of the cell can be studied.

These structures had been previously observed by Flemming and Benda (1882), but by less perfect methods. Nissl at first stained tissues hardened in alcohol with magenta red or methylene blue, and cleared in oil of origanum. This has been modified in several ways, so that at present his treatment is as follows: Small blocks of tissue are hardened in 96 per cent alcohol and fastened by Weigert's method with gum arabic, without imbedding. The sections are received in 96 per cent alcohol and stained in a watch glass. The stain is to be heated over the spirit flame until small bubbles arise which make a crackling noise (65° to 70° C.); sections are then transferred to aniline-oil-alcohol until differentiated. The process of differentiation is ended when no more coarse clouds of color go off into the fluid. The section is then transferred to the slide, dried with filter paper, after which some drops of oil of cajeput are applied, and the sections are again blotted with filter paper. A few drops of benzene are poured on the sections, blotted again, and finally some benzene-colophonium added, a cover glass placed over the section, and the slide heated until all the benzene gas has been driven off. Upon cooling, the section is thus permanently mounted.

The stain is made as follows: Methylene blue, B. pat., 3.75 grams; Venetian soap, 1.75 grams; distilled water or soft water, 1,000 c. c. The differentiating or decolorizing fluid has the following composition: Aniline oil, colorless, 10 parts; alcohol, 96 per cent, ninety parts. Nissl obtains his aniline oil directly from the factory at Höchst and keeps it carefully protected from the light. The benzene-colophonium is prepared by pouring benzene upon colophonium (white rosin) and allowing it to stand for from twenty-four to thirty hours. This results in a fluid transparent mass which can be brought to the desired thickness either by the

addition of more benzene or by allowing it to evaporate, and then it is ready for use. In mounting, while driving off the benzene gas by heating over the flame, the material may catch fire, but if the flames are blown out immediately no injury is done, and the alterations produced by the burning are quite characteristic and easily recognizable. The above description has been given in detail as it is the classical method, which, as above and with various modifications to be described later, has been used by the writer in this article. This method distinguishes within the cell-body always two and sometimes three constituents. One of these constituents of the protoplasm stains entirely blue by this method, and is spoken of by Nissl as the stainable or visible formed part of the nerve cell. The second part remains entirely unstained, and is spoken of by him as the unstainable or the visible unformed part of the nerve cell-body. In addition to the above, in many nerve cells, pigmentary deposits are visible which have been especially studied recently by Rosin (1896). Nissl has prepared a somewhat elaborate classification of cells according to the character and arrangement of the stainable portion to the non-stainable portion of the cell-body in different cells in different parts of the central nervous system. This stainable substance shows a series of different forms; smaller and larger granules of regular or irregular shape, arranged in groups or rows. These stainable masses are sometimes arranged in threads, smooth or rough, and varying in thickness, course, and length. Often larger structures, regularly or irregularly shaped, are seen, which stain with varying degrees of intensity, some appearing homogeneous, others showing a complex internal construction difficult to describe.

Three varieties of these larger bodies are especially to be noted, (1) the so-called "nuclear caps" (*Kernkappen*), stainable masses in the form of regular or irregular cones hollowed out like a cap and corresponding to one pole of the nucleus upon which it rests. There may be two such caps within one cell-body at opposite poles, and occasionally, according to Nissl, three such caps may exist within a single cell. (2) So-called "wedges of division" (*Verzweigungs-Kegeln*), stainable masses completely filling the angle at the point of division of a nerve cell process.

(3) Spindles, oblong spindle-shaped stainable bodies, thick in the middle and thinner at both ends, the latter sometimes tapering off into thread-like filaments. These spindles may be one or two-sided. Any one of these forms may be vacuolated. Nissl makes a rather elaborate classification of these ganglion cells according to their internal morphology, after spending years in the most exact investigations of various nerve cells in the different nerve centers of man and animals. He has come to the conclusion that definite types or variations of nerve cells exist which are constant not only in the same animal but often exist characteristically in homologous localities in a whole series of animals. According to him, all the cells in the nerve centers, except the so-called chromophile nerve cells, can be divided into two main classes. The first class includes the nerve cells which possess a well marked cell-body surrounding the nucleus completely on all sides, the protoplasm having a distinct contour. These are called somatochrome nerve cells. The second class (sub-divided into two groups, cytochrome and caryochrome) includes those cells in which, in Nissl preparations, the nucleus is most in evidence, the nucleus has a clear contour, but only indications of a cell-body are present, an appearance due to either the scanty development of the cell-body or to the predominance in it of the unstainable substance.

These cells often look as if they were naked nuclei, though by Golgi's method it can be shown that they may possess definite axones and dendrites. In some of these cells the stainable substance may be present, but very unevenly distributed, at different points in the cell, the nucleus appearing as if only partially surrounded by protoplasm. Nissl classifies these cells with such an ill-defined cell-body, the nucleus appearing only partially surrounded and not exceeding in size the nucleus of a neuroglia cell or of an ordinary leucocyte, as "granules" (Körner) or cytochrome nerve cells. These cells are present in great numbers in the granular layer of the cerebellum, also in the cerebral cortex and the olfactory bulb—being different varieties and not identical. The second subgroup of cells, in which the cell-body is only indicated but the nucleus is of the size of an ordinary nerve cell, or at least larger than that of a neuroglia cell, he designates as

caryochrome nerve cells. There are varieties of these as in the substantia gelatinosa Rolando, and those of the ganglion habenula—these types being designated at present by letters of the Greek alphabet. A majority of the nerve cells fall in the first group—the somatochrome cells—where the cell-body, in the light of morphology, has apparently greater relative importance than the nucleus. In this group are a series of types of nerve cells to be distinguished from one another partly through differences in the nuclei, but chiefly through different relations of the stainable and unstainable constituents of the cell-body.

These somatochrome cells were originally divided by Nissl into four groups, but now he includes them all in three groups as follows:

Group I. Arkyochrome nerve cells—stainable portions of cell-body in Nissl preparations in the form of a network, branches of which appear to be distinctly connected. Nissl notes, however, that in many of these cells processes can be made out into which the distinct network of the perinuclear part of the cell-body can go over, thus forming a parallel-striped arrangement. These arkyochrome cells Nissl subdivides into enarkyochrome forms and ampharkyochrome forms. The former shows the stained constituents arranged in the form of a network, which differs from the latter in that the intensely stained radiating nodal points of the network are connected in the cell-body by deeply stained thick bridges so that a further connected network of very deeply stainable substances is observable. Both these sub-groups of cells are widely distributed throughout the central nervous system. The former have been observed by Nissl in the spinal cord, but are most numerous in the large dorsal nucleus at the proximal end of the medulla. What he formerly classified as Group III, arkyostichochrome nerve cells, are now included in this first group (1897a). He describes these cells as presenting a striated appearance with a network-like structure united in a most intricate manner.

The Purkinje cells of the cerebellar cortex were given as typical examples of cells of this sort. (See Plate IX, Fig. 27).

Group II. Stichochrome nerve cells, stainable substance ar-

ranged in striæ, running in same direction, and usually parallel to contour of cell-body, in part also with surface of nucleus. These striæ, made up of different stained elements, threads, spindles and granules, are more or less isolated and in rows. Nissl describes four types of these cells represented by (1) nerve cells of motor nuclei, (2) large cells of cornu Ammonis, (3) certain cells of cerebral cortex, and (4) certain cells of spinal ganglia.

Group III. Gryochrome nerve cells, stainable substance entirely made up of small granules distributed in threads or heaps. Nissl does not give an illustration of these cells, but states that they are to be found particularly but not exclusively in the corpus striatum. Nissl states further that transitional forms exist which are difficult to definitely classify, but that the cells of a wholly definite structure are situated throughout the animal series in homologous localities. He has also shown that all these types of cells may, under certain circumstances, show different staining relations (1894a), that is, the individual members of a certain group of cells belonging to one type may be palely, moderately, or intensely stained. This difference seems to depend upon the concentration of the stainable substance in the cell-body. The darkly stained cells he designates as pyknomorphous cells, in which stainable portions are arranged relatively most closely; intermediate stages are designated as parapyknomorphous cells; while very feebly stained cells are designated as apyknomorphous, in which stainable masses are not arranged close to one another but are somewhat widely separated by the non-stainable constituents of the cell-body. Nissl furthermore mentions that often the nucleus shows modifications which correspond in greater or less degree to the staining intensity of the cell-body, so that in an apyknomorphous cell the unstained nuclear substance is relatively more abundant than in the pyknomorphous cells. This holds in the somatochrome cells, and to a less degree in the caryochrome and cytochrome cells.

Supposed to be more or less in the nature of an artefact are the so-called chromophile nerve cells which one finds often single or in small groups along with the other nerve cells, but in which the stainable substance seems to be evenly diffused throughout

the cell-body, making it impossible to distinguish a stainable from an unstainable constituent in the cell. Nissl (1896c) states that they are always relatively smaller than pyknomorphous cells. The explanation of these forms is not as yet entirely satisfactory, but they are supposed to be due to the action of the reagents employed, though under certain circumstances they may have a pathological significance. Another nomenclature has been introduced by Flesch (1897), in which he speaks of chromophilic cells and chromophobic cells with transitional forms, and attributes the differences to variations in the internal chemistry of the cells, this latter depending in part upon differences in the development, in part upon differences in metabolism or function.

Von Lenhossek (1895) gives a very accurate description of the appearances within the cells of the ventral horn of the spinal cord and in the cells of the spinal ganglia. He has found that thionin stains as well if not better than methylene blue, and Barker (1899) has obtained similar results, but states that crystalline deposits have been more frequent in preparations stained with thionin than in those stained with methylene blue. In material hardened in Lang's solution or other corrosive sublimate solutions, the writer has at times detected crystalline deposits, whether stained with thionin, methylene blue or methylene violet, and attributed the same to the fixative fluid and not to the stain at all, as no crystalline deposits have been observed with other fixative agents—alcohol, formalin, and Van Gehuchten's fluid—regardless of which stain was used.

It would be interesting to know whether corrosive sublimate fixation was used by Barker in the above mentioned preparations. Van Gieson uses polychrome to a considerable extent as a stain, but it seems to the writer to give a paler and less distinct stain than either methylene blue or methylene violet, and has a tendency to fade after a time. Referring to von Lenhossek again, he objects to the term "granules" for the stainable substance, the masses being ordinarily much too coarse to be so designated. He has given accurate descriptions of their characteristics and arrangement in various animal species. This stainable substance of Nissl he designates as "chromophile corpuscles," and later uses the term "tigroid masses" (1896).

De Quervain (1893) has suggested that the chromophilic bodies or corpuscles are aggregations of fine granules, but von Lenhossek refuses to admit that all such bodies represent aggregations of minute granules. Schaffer (1893) first described the peculiar behavior of the axone and adjacent portion of the cell-body known as the axone hillock as regards Nissl staining, this space being entirely free from this stainable substance of Nissl. With Kronthal's method the axone and axone hillock stain intensely in methylene blue, but Benda (1895) found that when specimens thus prepared were cleared in creosote this region lost its color and only the stainable substance of Nissl in the cell-body and dendrites retained its color. He found one exception, however. In the basal axones of the pyramidal cells of the cerebrum, especially of those known as the giant pyramidal cells of Betz, just at the beginning where a collateral is coming off at right angles, a small wedge-shaped granule, triangular in section, takes up the methylene blue, the axone itself not staining at all. These different results obtained by differences in technique emphasize the writer's previous statement of the necessity of a full knowledge of the history of the case and every detail of technique before coming to any conclusions in a given case.

With this brief review of the more important literature on the internal structure of the nerve cell we will now turn to the arrangement of the cells in the cortex. To go into the literature of this subject in detail would be sufficient material for an article in itself, so that it will be passed over as briefly as possible. Table I shows the arrangement of the cells as described by the more important contributors to this subject. As early as 1782 Gennari divided the cortex at the posterior pole of the cerebrum into three layers, an external and internal gray layer separated by a white layer (so-called line or band), the "line of Gennari." Vicq d'Azyr, in 1786, described this region as also made up of an inner and outer gray layer between which was a white layer (line or band), the "line of Vicq d'Azyr." Meckel, in 1812, stated that the gray layer of the cortex is single in all parts but the occipital region and cornu Ammonis. In the occipital region a band of white substance, the line of Vicq d'Azyr, separates the gray substance into an

external and internal gray layer, so that the structure of this part of the brain is more complex than the rest of the external cortical region. Coming down to 1840 Baillarger first examined the cortex carefully, and described three gray and three white layers, or six layers in all, alternating gray and white. From his descriptions the line of Gennari or Vicq d'Azyr, in the occipital region, received the additional name of the "line of Baillarger." In 1841 Remak examined the cortex microscopically as well as macroscopically, but without the use of staining agents, and described a four-layered type made up of two gray and two white layers alternating with one another.

Somewhat later (1852) Kölliker described a three-layered plan, the inner layer appearing of a yellowish red color, the middle of a grayish color, and the outer of a whitish color.

Berlin, in 1858, first used stains in his work, hardening pieces of the cortex in chrome salt and staining with carmine. He described six layers as the type, the upper five from the character and arrangement of the cells and the lower as a layer free from cells. He describes the layers from within outwards toward the surface, reversing the usual order of all other authors, and leading to considerable confusion, unless this fact is carefully noted. Clarke, in 1863, described eight layers as the common type, and Luys the following year, 1864, described just one half that number, or four layers, as the common type. Arndt, in 1867, described a five-layered type, and Meynert in the same year divided the cortex into a motor and a sensory region, the former anterior to the fissure of Rolando, and the latter posterior to the same. He described five layers as the common type, and eight layers as the type in the occipital region. Cleland, in 1870, described rather a complex arrangement of the cortex into a four-layered type, the third layer being separated from the fourth by what he designated as the "primary pale band." The "deep pale band" is included in and forms the lower part of the fourth layer. Henle in the same year described a four-layered type made up of (1) the outer layer, (2) outer spherical cell layer, (3) pyramidal cell layer, and (4) inner spherical cell layer. Charcot, the following year, 1871, described the layers of the cortex as five in

number, (1) superficial layer, (2) small pyramidal cell layer, (3) larger pyramidal cell layer, (4) granular cell layer, and (5) fusiform cell layer; approximating that used by many of the subsequent authors. Lewis, in 1876, described five layers as the type for the motor area and six layers as the type for the sensory area. Major in the same year, 1876, described six layers as the common type, and Krause, also in the same year, described seven layers as the common type. Betz, in 1881, made the division into five layers as the type for the motor area, but divided the sensory region into eight layers as the type—a classification somewhat similar to Meynert's, but with a different nomenclature.

Golgi, in 1883, using his impregnation method, divided the cortex into three layers as the type, (1) superficial or small pyramidal cell layer, (2) middle or larger pyramidal cell layer, and (3) internal or irregular cell layer. Schwalbe, in 1885, described a curious arrangement of the cortex into four layers, the upper two known as the "outer Hauptzone" or chief zone, and separated from the lower into two layers, known as the "inner Hauptzone" or chief zone by the "boundary zone" or "line of Baillarger." Obersteiner's (1887) division into a five-layered type is similar, except as to the nomenclature, to that of Charcot's common type, Lewis' motor type, and Betz's motor type. Cajal, in 1890, recognized the same three layers as Golgi in the common type, but added a fourth layer, which he called the first or molecular zone, then followed the second, or zone of small pyramidal cells; the third, or zone of large pyramidal cells, and the fourth, or zone of polymorphous cells; thus making four layers in all. Golgi's first layer is thus seen to include the first and second layers of Cajal. In the occipital lobe, Cajal describes an additional layer of vertical fusiform cells between the first or molecular layer and the layer of small pyramidal cells, the other layers being similar to his common type, and thus making five layers in this region. Gowers, in 1893, adopted practically the same division into a five-layered motor type and six-layered sensory type as described by Bevan Lewis in 1876. Hammarberg, in 1895, described six layers as the type, with numerous variations in different parts of the cortex. Edinger, in 1896, describes

four layers, similar to Cajal as the type, and Starr (1896), in his atlas of nerve cells, also describes a similar arrangement. Nissl (1898) describes a four-layered type as follows: First, or layer poor in cells; second, or layer of pyramidal cells containing 2 equals layer of small pyramidal cells (equals 2 of Meynert's layers) plus 3 equals layer of larger pyramidal cells (equals 3 of Meynert's layers); third, or layer of small cells (equals 4 of Meynert's layers); and fourth, or internal (6) plus external (5) zone of the layer of medullated fibers (equals 5 of Meynert's layers). Region 5 equals ganglion cell layer, and region 6 equals spindle cell layer of Hammarburg. It will be seen that there is much variation and not a little confusion in adjusting these various arrangements and classifications to one another, and in order to simplify the matter as much as possible the writer suggests that as the large and small pyramidal cells are so intermingled and merged into one another, they should be included in one layer, thus making three layers the common type, as follows: First, or superficial layer, corresponding to the first layer of Cajal and Edinger; second, or pyramidal cell layer, corresponding to the second and third layers of Cajal and Edinger; and third, or spindle cell layer, similar to the fourth layer of Cajal and Edinger. This arrangement differs from Golgi's three-layered type in that the latter recognized no superficial or tangential fiber layer, but includes this as the upper part of his first, or layer of small pyramidal cells, whereas in the writer's arrangement one pyramidal cell layer includes both the large and small pyramidal cell layers of Golgi. The third layer of Golgi and of the writer are the same as the fourth layer of Cajal and Edinger. This classification into three layers as the type will be used for convenience in the subsequent description of the plates. That there are variations from the type in different parts of the cortex, of course, is recognized by all who have worked in this field to any extent, and, furthermore, specialized parts of the cortex have a specialized arrangement, as the cornu Ammonis, for example.

In turning now to the pathological investigations by means of the Nissl stain (1898), they are found to have been exhaustive and varied, including most parts of the central nervous system

in both man and animals and in many forms of disease. A brief review only will be given of the contributions containing descriptions of researches by Nissl's stain in that form of mental derangement known as dementia paralytica, or general paresis. Nagy (1894) carried on his investigations by means of the Nissl stain, and found the greatest changes in dementia paralytica, including various stages of cell degeneration up to complete destruction of the same—the cells most altered being those of the frontal lobes, and least changed those of the occipital lobes. Changes of a high grade were also shown in the cells of cases dying after severe epileptic insanity—here the gyrus rectus and cornu Ammonis being affected the most. In chronic forms of insanity similar changes were found, but the number of cells entirely destroyed was undoubtedly smaller than in the above mentioned forms of illness. In acute hallucinatory confusional insanity only the beginning stages of alteration were found; similarly in mania. He finally states that each form of mental derangement showed the highest grade of change in which the clinical picture of the worst suffering was present, while in the curable forms there were found corresponding slight changes. Belmondo (1896) employed the Nissl method in investigating the alterations in the nerve cells in dementia paralytica, and did not find changes of great gravity—at most the cell protoplasm being much diminished and disintegrated; now and then pigmentary atrophy, as much in the Rolandic region as the frontal lobe, is found, and in other parts a diffuse chromatolysis is to be seen. He condemns the expression meningo-peri-encephalitis, which implies a conception of an inflammatory process.

Boedecker and Juliusburger (1897) examined sections of cortex from the central and parietal convolutions of three cases of dementia paralytica by the Nissl method. They found thickening of the pia with septa projecting into the cortex, containing blood vessels whose walls showed no special thickening but were surrounded by rich deposits of pigment granules, which latter were also found here and there, distant from the septa in the cortical network. In regions most affected the cortex did not present its well known layering. There was to be seen a thickly-

compressed granular crowding with different shaped granules; thin, spindle-shaped, round, or oval, with strongly colored ones lying adjacent to those only slightly colored. There was increase of blood vessels and hypertrophy of the interstitial network, with corresponding decrease in the number and size of the cells. Many cells were considerably diminished in size, markedly shrunken, entirely without processes, and intensely colored. At times a differentiation into nucleus and granular cell-body was not possible, owing to complete chromatolysis, and very seldom were cells found with a strongly colored nucleus, strongly colored fine granules about the same, with larger granules at the periphery of the cell-body—a partial chromatolysis.

By the Marchi method these investigators determined a degeneration of the fibers from these cells extending into the spinal cord. They conclude the process to be an intense degeneration and proliferating one, going on hand in hand—degeneration of the cells and proliferation of the interstitial network, with increase of blood vessels. Crisafulli (1897) notes a great variety of changes in dementia paralytica. The cellular changes are most advanced and diffuse in the frontal lobes, but are not limited to that region. He examined sections from the frontal, parietal, occipital and temporo-sphenoidal lobes of both hemispheres. He found pallor, granular disintegration, and loss of chromatic substance. Often the cell-bodies were atrophied or contained an excess of yellowish pigment, and their numbers were reduced. The nuclei were often eccentric, and all stages of the destruction of the nucleus were observed. While the alterations shown by Nissl's method were not less constant than those demonstratable by other methods, Crisafulli does not consider them characteristic of the disease or in any way different from those seen in some other mental diseases. They are, however, more or less grave and diffuse and not limited to a single cortical center; with varying degrees of degeneration of the nerve cells. He further states that, provided the nerve cell element degeneration is not greatly advanced, it is impossible to find any alteration of the blood vessels, and, finally, that when the psychosomatic condition of the paralytic is not greatly aggravated and death intervenes from

other causes, it is possible that the pathological report will note some elements which are not degenerated, although there may be various alterations. His article is illustrated by eight figures showing cells in various conditions of degeneration.

Angladi (1898) reports a case of acute dementia paralytica in which death occurred after a series of epileptiform convulsions when the patient was at the age of thirty-seven years. The autopsy showed peri-encephalitis. Preparations were made from the ascending frontal convolutions and the anterior part of the frontal lobe of the left hemisphere. He states that not a single one of the pyramidal cells preserved its normal characteristics, and the transformation in the great majority of cases seemed to be various stages of the same process—various stages of chromatolysis, vacuolization, eccentricity of the nucleus up to rupture of the cell wall and extrusion of the nucleus and, finally, complete destruction and disappearance of the cell. The chromatic substance is first attacked, and some few cells relatively healthy showed about the nucleus the first stages of dissolution. Destruction of the achromatic network was shown by formation of vacuoles at the periphery of the cell, increasing gradually up to complete destruction of the substance of the cell. The nucleolus becomes vacuolated and disappears. The nucleus is attacked by central chromatolysis and disappears *in situ*, or becomes eccentric, or the cell-body may rupture and the nucleus be extruded, becoming irregular, compressed or shriveled. The contour of the cells is always irregular, and the prolongations either broken or tortuous. He states that the cells of the medulla and cord show identical alterations. Angladi finally concludes by stating that we do not know whether these lesions are the cause or the result of the malady, and asks the question, "Are the lesions primary or secondary to an alteration of the vessels or interstitial tissue?"

Berger (1898) examined the anterior horn cells of the spinal cord in twelve cases of dementia paralytica and found lesions affecting principally the chromatic substance in 83 per cent of the cases. He failed to find a strict parallel between these cellular lesions and those of the fibers and cortex, or between them

and the clinical symptoms of the disease. He illustrates his article by figures of these cells.

Nissl (96a), in 1896, stated that he maintains the same position as Kraepelin, namely, that one sees in dementia paralytica a general disease with the histopathology directed especially to the cortex. The pathological changes in the blood vessels are obscure, and the relation of the glia to the blood vessels is complicated, also the condition of the lymphatic vessels. Some authors regard the disease as an inflammatory process, others that a chronic interstitial inflammation enters into it, others that it is a parenchymatous process, and still others that it is a histopathological process in which the specific tissue is diseased primarily. Nissl notes that an inflammation without the blood vessels sharing in it cannot be thought of. He states that the appearance of the paralytic cortical disease can be present without the blood vessels being diseased and without the blood vessels containing any elements of an inflammatory process. Also there may be a high grade of disease of the tissue (*Gewebe*) in excess of any disease of the blood vessels, and only a slight involvement of the tissue, and *vice versa*, a severe tissue damage with only insignificant disease of the blood vessels. Also direct inflammatory blood vessel changes with infiltration of the walls of the same with leucocytes and "mastzellen," which may rarely pass out into the adjacent regions. In some cases this inflammatory alteration of blood vessels results in a massive production of decay of numerous neurones. These inflammatory changes in the blood vessels have nothing directly to do with the chief paralytic process, and are only found in the cortex when sepsis-producing bacteria are present. It therefore follows that the paralytic cortical disease can be regarded either as the result of disease of the blood vessels or as an inflammatory process. Almost all cases of dementia paralytica show a slight or severe disease of the blood vessels. There may be also a leptomeningitis, disease of the beginning part of the aorta, an injury to the diploe, brittleness of the bones, or there may be a general arterio-sclerosis. If it is not a disease of the arteries or an inflammatory process, it may be a primary disease of the glia or of the cortical neurone. Changes in glia

are progressive in kind (mitosis of glia nuclei, hypertrophy of glia cells, and increase of glia fibers) with regressive changes in the cortical neurone—an acceptance that results of investigation directly contradict, since we find in the most luxuriant increase of glia that the nerve cells are only slightly or not at all changed, and *vice versa*, in the most severe nerve cell changes the increase of glia may be only slight. The acceptance of a primary glia fiber increase is absurd, since the glia fibers are an intercellular substance. Nissl therefore concludes that the cortical disease of dementia paralytica is a primary disease of the cortical neurone; at the same time with the regressive changes in the cortical neurone goes the progressive changes in the glia cells. Histo-pathological investigations of paralytic cortical disease has to deal chiefly with the following difficulties: (1) a paralytic diseased cortex and the, to us, available cortex of the dead paralytic are two different things. Entire series of original circumstances that damage the neurones of the cortex without having anything to do directly with the paralytic process is to be noted, as the complete closure of a blood vessel which only indirectly bears upon the original process. Also death of a paretic from typhus fever, septic pyemia, etc. The problem is extremely difficult, as there is no specific disease of the cortical cells, and, furthermore, there is no paralytic cortical cell disease, although there is a paralytic cortical disease. The kind of disease of the cortical tissue is worthy of being pointed out. (2) It is not sufficient to know that the cortical neurone is diseased, but it is important to know which cortical neurones are damaged. Attention to the kinds of nerve cells directly diseased is important for the real conception of the disease process, and also for the critical examination of the plan of the cortex and its functions. Nissl differentiates the following forms of disease of the cells of the cortex in dementia paralytica: (1) Acute progressive disease. In certain cases the disease ends with the complete destruction of the elements of the cortex. (2) The chronic disease progresses slowly, resulting in either a pigmentary degeneration or in a decay of the cell-body and nucleus, and ends with so-called cell sclerosis. (3) The severe disease of the cortex which runs either an acute or sub-

acute course and terminates with the death of the cell. The necrotic cells persist commonly either bleached out or having the appearance of colliquation or of vacuolization of the ground work. (4) The combined form of the disease, in which the cell may be acutely diseased without either a cure following, or, on the other hand, the ordinary course being taken that terminates with the destruction of the cell. Midway in its course the disease process is arrested and takes on the symptoms of the chronic disease. The severe cortical cell disease is entirely overlooked by former authors. It differs in that the nucleus is also involved and a process of liquefaction takes place in the same. It becomes smaller, shrunken, the contours become homogeneous and tinged, the nucleolus sinks to the nuclear wall, which latter is irregular and shriveled into folds, the network cannot be distinguished, and vacuoles and crystals may be formed. In the necrotic cells calcareous deposits may be found, as is seen in other cortical cell diseases, as sclerotic elements. These chalky or calcareous concretions occur in the form of fine granules, crumbs, plaques, or stalactitic masses, which are intensely colored with methylene blue. The entire cell may be bleached or only a single part, as some of the fine dendrites or only a single dendritic process, or only the nucleus, or, finally, only the nuclear membrane. This calcification, moreover, is an exceedingly important phenomena, since we are entitled to conclude that partially calcified cells are no longer functionally active and are necrotic. Under similar conditions in acute diseases we recognize the phenomenon of death in the affected cells. If one places the preparations in alcohol after twelve hours postmortem, any mistake in this direction may be obviated. Whoever grasps the histopathology of the cortical cells will guard against any mistake in regard to the above mentioned death phenomena. Ewing, in 1898, used the Nissl method in observations upon the changes found in ganglion cells of the central nervous system in various pathological conditions, and stated that various grades of chromatolysis were found in the cortical cells in dementia paralytica.

Ballet (1898) exhibited sections from the paracentral lobule of both a normal and a paretic brain, stained by the Nissl method.

In the normal brain, under a magnification of forty-five diameters, the four layers of Schwalbe and Ramon y Cajal were easily discoverable, but in the paretic sections, in marked contrast, they were recognized with difficulty; also in the latter, great numbers of capillaries were noted in the third layer and white subcortical substance, the nerve cells were less numerous, and in all cases less distinct and lost in the midst of a mass of nuclei. At 130 diameters the contrast was still more marked, and at 250 diameters vascular lesions were noted consisting of enormous dilatation of the capillaries and arterioles, the investment of these vessels by a casing of lymphatic corpuscles which distended the adventitious sheaths, and the accumulation of pigment at certain points, particularly in the neighborhood of the bifurcations. These alterations are also revealed by hematoxylin and picro-carmin, and have been described for a long time by all observers, but the Nissl method shows the changes more clearly than any of the others.

In addition to these changes one may mention the multiplication of the white corpuscles and their migration from the vessels by diapedesis, as due in part perhaps to the proliferation of cellular elements of the neuroglia which accumulate in the interstitial tissue, principally in the neighborhood of the vessels or about the nerve cells. At 600 diameters one easily distinguishes the small white cells (lymphocytes), with small nuclei deeply colored and with but a small amount of protoplasm; large white globules with protoplasm somewhat abundant and with voluminous nuclei irregular in form and less impregnated than those of the lymphocytes by the methylene blue; and, finally, polynuclear leucocytes. But interest in Nissl's method is chiefly in the study of the lesions of the nerve cells. At 600 diameters one sees the profound alterations undergone by these cells. In examining the elements of the third layer (large pyramidal cells) or the giant cells of Betz, there is a tendency in them to lose their triangular shape and become oval or rounded, the protoplasmic prolongations are atrophied and but slightly visible, the chromophilic granules for the most part undergo a process of disintegration and are reduced to a sort of fine powder or dust, or are entirely dissolved in the mass of protoplasm. The author then discusses the nature of the process,

some authors (Magnan, Mierzijewski, Mendel) claiming the primary disease to be that of the neuroglia framework—an interstitial encephalitis. Others on the contrary (Tuezeck, Ziegler, Binswanger, Joffroy, Pierret) claim it affects primarily the nervous tissue, either the nerve fibers or the nerve cells; thus being a parenchymatous encephalitis. The more Ballet studies the pathological anatomy of dementia paralytica the more he is convinced that the first and most important lesions are those of the blood vessels. These are seen in every case, while those of the cells are inconstant, variable in degree, and subordinated to those of the vessels. This is not to say that the cellular alterations may be, as some authors claim, the result of the mechanical choking of the cells by the proliferating interstitial tissue which makes their pathology more complex, but he thinks the result is due less to the compression by the thickened neuroglia than to the difficulties of assimilation, owing to either the circulatory obstruction or the action of the toxines carried by the blood. Ballet then discusses the relation of syphilis to dementia paralytica, and states that the pathological anatomical findings in the two cases are practically identical, and advocates a syphilitic etiology for the disease.

In this historical review of the literature on the pathological conditions found in dementia paralytica by the employment of the Nissl method, it is to be noted that but few of the contributions were accompanied by figures illustrating the various pathological findings described in the text.

(To be continued.)

CASES OF COMBINED MORPHINE AND ATROPINE POISONING.¹

BY PHILIP ZENNER, A.M., M.D.,

CINCINNATI.

These cases were deemed worthy of report because none like them, so far as I could learn, have been published. They seem to indicate that poisoning with the two drugs combined is especially dangerous, notwithstanding their supposed antagonism.

The first case was reported at the time of its occurrence some years ago. The patient was a nurse, of twenty years, who had taken with suicidal intent, hypodermic tablets each containing one quarter of a grain morphine, and 1-150 grain atropine. Nothing was known of this at the time, and it was only discovered hours afterwards when, after a careful search, some tablets were found under her pillow. It was supposed, judging from the number of tablets remaining in the bottle wherein they were contained, that she had taken about eight of them, but that may have been far from the mark.

She was found to be unconscious at 6 P. M. I saw her a few hours later. At that time she was profoundly unconscious. There was complete flaccid paralysis of all the extremities. The face was expressionless, eyes slightly divergent, the pupils of medium size and irresponsive to light. Pulse was 120 per minute, of fair force and volume. Temperature 100. Respiration eleven per minute, and very irregular. Marked cyanosis, knee-jerks could not be elicited. Urine contained no albumin, specific gravity 1,012.

All efforts at resuscitation were futile—washing out the stomach, introduction of coffee through the stomach tube, hypodermic injections of whiskey and ether, applications of the electric brush, etc. The breathing was the ominous feature throughout, quiet, never stertorous, but irregular, the interval between the respirations becoming gradually longer and longer. The pulse, which was good, though rapid, throughout, could be felt for some time

¹Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

after the breathing had ceased. The pupils gradually became more dilated, though never to the degree of full dilatation. She died at 3.30 A. M.

The second case was a young girl of thirteen years, who had chorea for about two weeks. Her restlessness was so great that the physician left for her some hypodermic tablets, each containing morphine gr. one-eighth, and atropine gr. 1-200. She received of these tablets one at 8 P. M., one at 9 P. M., one at 10 P. M., one at 11 P. M., one at 11.30 P. M., and one at 12 P. M.; that is, six tablets within four hours. She fell asleep an hour or two after the last tablet had been taken, and slept soundly until 6 A. M., since which time she was dozing and waking, and when awake delirious, until I saw her. I made a hurried call about 11.30 A. M., the only time I ever saw her. She seemed asleep when I stepped into the room, but soon awoke, and was quite restless. At times she would squirm about and scream, "Oh, that cramp," doubtless on account of abdominal pain. She gave sufficient answers to questions to indicate that she was fairly conscious, those in attendance stating that she was now more lucid than she had been at any time during the day. Her temperature was 104° F. Pulse was rapid and thready. Scarcely to be counted, but about 130. She was very cyanotic. Her heart could not be carefully examined, and her restlessness prevented a satisfactory examination of reflexes. She had vomited everything she had taken that day.

As nearly twelve hours had passed since taking the last of the atrophine and morphine and her mind was becoming more lucid, I thought the prospects for recovery fair, even though her condition appeared critical. Nevertheless I telephoned at once to her physician, telling him she ought to be stimulated and carefully watched. A few hours later he said her pulse was fair, and he thought she was doing well. He did not see her again. Perhaps she was doing well, but about 8 P. M. she was seized with severe convulsions and died within an hour. I have little definite information of her condition subsequent to the time of my visit.

I saw so little of this second case that I can scarcely make a comparison of the two cases. The most striking difference was

in the pulse, which was very good in the one and bad in the other. That may have been due to the fact that the proportion of atropine was much larger in the latter.

I know of but two reported cases of a somewhat similar character, and these I mentioned in my former report.

The first was that of a man of sixty, who took by mistake a tablespoonful of a mixture containing equal parts of liniment of belladonna and tincture of opium. Two and one half hours after swallowing the mixture he was unconscious, pulse 140, breathing rapid, pupils dilated and sluggish. Twenty-four hours later he appeared to be out of danger. Four hours subsequently he sat up in bed to take a drink and fell back dead.

The second case was a woman of seventy-nine, who took by mistake ten grains extract belladonna, ten grains of extract of conium, and eighty minims of tincture of opium. Two hours later she lay in profound coma, breathing hurried and noisy, pupils dilated, pulse 130, feeble and intermittent. The patient was resuscitated to the extent that she took nourishment fairly well, but she died forty-eight hours subsequently of exhaustion.

These two cases are, perhaps, not to be compared with my own, as age and enfeeblement may have led to the fatal termination. But nevertheless so far as they go, they seem to lend support to the view already expressed, that poisoning with a combination of the two drugs is peculiarly dangerous.

A NOTE ON PERIODIC INSANITIES WITH REPORT OF THREE CASES OF INTERMITTENT MELANCHOLIA.¹

BY ALFRED GORDON, M.D.,

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PHILADELPHIA, PA.

The periodicity of certain forms of insanity has not been frequently the subject of a special study by the alienists. The reason of it lies probably in the fact that cases of this nature are rarely met with in institutions. Yet, outside of asylums, they are not as rare as it may appear.

The writer has had the opportunity to have under his personal observation quite a number of cases in and outside of asylums. A close study of them leads him to consider the subject of sufficient importance from both the scientific and practical points of view, so that he considers himself warranted to report the result of his observations.

Under the term Periodic Psychoses we understand attacks of mental derangement occurring at certain intervals, during which the patient is more or less free from mental disturbances. Some alienists, especially the older ones, maintain that the intervals between the attacks must be of equal duration in order that the psychosis be called periodic insanity. The reading of the records, however, shows that the duration of the intervals and of the attacks of the psychoses are not at all characteristic features of periodic insanities. Their duration varies.

The periodic psychoses known are mania, melancholia, and paranoia. Krafft-Ebing and Kraepelin also include the circular insanities.

While mania is more likely to occur periodically than the other forms, melancholia is by no means infrequent. The reason why the periodicity is often overlooked is that it is not easy to keep this class of patients under observation a sufficient length of time.

¹Read before the Philadelphia Neurological Society, April 28, 1903.

Among all the cases that came under my observation, there are three that I have had the good fortune to have under my care for the last four years, and thus have had an unusually good opportunity to follow the individual attacks step by step and keep complete records. A close study of them showed me the remarkable uniformity in the character of the onset of the attacks, in the character of the termination of the attacks, in the age, in the family history, and in the fact that each subsequent attack was more and more pronounced. A still closer study will make us see clearly that in patients presenting the periodic forms of insanities, there is undoubtedly an unusually pronounced degenerative basis, upon which are built these occasional outbreaks of depression with or without delusions. The histories of the cases are as follows:

Mrs. E. O., aged thirty-five, was seven times pregnant. Four years ago, after her fourth confinement, in the midst of apparently perfect health, she became suddenly depressed and felt she could not do her work. As soon as she commenced to work she imagined she could not continue because she feared not to be able to do it properly. Soon her worriment increased, because she could not do anything for her children, whom she loved very dearly. She used to prepare the food for them, and now she found herself incapable of doing it. Conscious of this condition, the patient soon reached a state of complete discouragement. She ceased to take an interest in her household, in her husband and children. The painful moral depression became associated with the idea of physical and moral impotence which showed itself in the lack of confidence in herself and in the absence of initiative and determination. The patient's life was dominated by sad ideas; she could not take pleasure in anything; everything and everybody became to her a source of constant irritation and suffering. Tortured by the complete inability to accomplish anything and to render the slightest service to her family, to whom she was previously a devoted mother and wife, she could not understand why she suffered so much. She then arrived at the conclusion that she was a worthless being condemned to perish. At the same time the struggle between the state of consciousness of this miserable existence and the inability to accomplish anything put the patient in a state of constant anxiety which had its effect also upon her vegetative life. She lost totally her appetite and would never ask for food; her digestion suffered considerably. The special senses became also affected; the taste and smell were so perverted that she rejected

almost every article of food with disgust. It was astonishing to hear the husband telling how small was the quantity of food the patient received: Only two or three times a day she would take a spoonful of milk and a couple of slices of bread. She soon began to lose in weight. The respiration was slow and superficial. The pulse was small, filiform and accelerated. Pain in the precordial region was frequent. The blood examination at different periods showed a condition similar to that of chlorosis with the exception of a marked leucocytosis (22,000 per ccm.).

If we add to these symptoms headache, partial insomnia, with frightful dreams and occasional visual hallucinations, the picture of the patient's condition will be complete.

The mental state just described remained almost unchanged for two months. Only now and then a ray of hope would make its appearance. She would cease to groan and moan, complain less and speak less of self condemnation; then the somatic symptoms would become ameliorated. These slight remissions would last only a few hours.

At the expiration of two months, one morning the patient awoke in a calm and peaceful manner, called her children, began to caress them and offered to prepare breakfast for the family. The mental faculties became clear and normal. She began to take an interest in her surroundings, in her own personality; briefly speaking, the melancholic state by which she was tortured for two whole months disappeared entirely.

She became pregnant again and felt well during ten months. Three weeks after the confinement, which was normal, suddenly, and without any preliminary symptom, the old mental trouble returned. Again the same complaint of worthlessness, the same indifference to her surroundings, again the same state of anxiety which made her this time speak of suicide. The knowledge of the fact that this was her second attack made her fear that her case was lost, that she would never get well, and therefore the symptoms of anxiety and the somatic symptoms were more pronounced. The visual, also auditory, hallucinations occurred very frequently. This state of depression lasted two months. Exactly like in the first attack, this second one also terminated abruptly, and again she became normal. A third pregnancy occurred. Six weeks after the confinement the old trouble returned. Without any prodromal symptom she became markedly depressed. The self condemnation with the delusions of worthlessness, also the tendency of suicide, hallucinations and the painful emotional state recurred.

Shortly speaking, the patient passed through a state of mental disturbances almost identical in its evolution with the first two attacks, with this difference, however, that the last attack lasted

four months, while the first two attacks lasted only two months. The termination was also abrupt.

The patient is now perfectly well. A year elapsed since the last attack. She is not pregnant.

In her family history I could elicit two cases of insanity—in a sister and grandmother. She had eight children, two of whom died with hydrocephalus, one died immediately after birth; one of diphtheria.

Her personal previous history shows that she suffered from obsessions at the age of sixteen, and was always considered highly irritable.

Case 2. Mrs. B. B., aged thirty-one, came under my observation three years ago with a typical melancholia. Delusions of unpardonable sin of a religious character, hallucinations, both visual and aural, and an array of neurasthenoid symptoms. All these symptoms were present at the time I first saw her. From the previous history of the case I could detect one previous attack, which was evidently mild in character, but was undoubtedly present. The second attack, in which she came under my care, was sudden, without a preceding physical indisposition or any mental disturbance. As a woman of means, she enjoyed life, and was mentally lucid. One morning, after having spent the previous night in a theater, she awoke in a state of depression, and from that hour on the above-mentioned symptoms gradually developed.

A peculiar fact to note is that this attack, as well as the first, which I did not witness, began with a facial neuralgia which lasted only one day.

The condition lasted three months. The termination of the disease was strikingly abrupt. Six months later, apparently without any cause, the patient again became depressed. Here again the above mentioned neuralgia was present on the first day of the attack and then gradually disappeared. All the symptoms of the previous attack repeated themselves, but they became more pronounced. The hallucinations tormented her considerably, so that the patient made repeated attempts at suicide. Equally the somatic symptoms were of a more marked character. The management of this attack was also more difficult than that of the first attack. The duration was also longer. It took six months for her to recover, but the termination was as abrupt as in the previous attack.

The family history shows that all her sisters are of a highly nervous temperament. An uncle died of parietic dementia and a grandmother died in an insane asylum. At the age of fourteen the patient presented for a short period symptoms of obsessions and agoraphobia, and at fifteen chorea twice during the same year.

Case 3. Mrs. R. P., aged thirty-four, had two attacks of

melancholia, before she came under my care. Her husband, a man of intelligence, gives a clear account of her previous illness. After a confinement she developed a phlebitis, from which she recovered at the end of six weeks. During convalescence she suddenly became depressed. The condition grew worse and worse, so that she finally presented distinct symptoms of simple melancholia without delusions. The prominent symptoms were a pronounced depression, and indifference to the surroundings; the patient would spend her time in groaning and moaning, and lost interest in everything. The first attack lasted six weeks, after which the recovery came on abruptly and unexpectedly.

Six months later a similar attack occurred without any preliminary illness. The character of it was exactly like that of the first, but it lasted three months. Again the patient recovered suddenly.

I have had her under my observation for the last three years, during which time she had two attacks. The first of the latter also came on in the midst of apparently perfect health. Besides the painful emotional state, she presented also delusions of personal worthlessness and self condemnation, which at times led her to attempts at suicide. The condition, which lasted six months, was decidedly of a more serious nature than during the first two attacks.

After a recovery, which set in also unexpectedly, the patient remained in good health for eight months. After this interval of well being, another attack occurred.

This time she developed within a very short period a typical melancholia, with delusions of unpardonable sin; her sufferings, she says, are all due to her being untrue to the Lord. Her retribution, she says, is just and she must die for it. She is also tortured by frightful dreams, in which she sees her own body cut and burned by some invisible agents in punishment for her lack of religious ardor. There is no use, she says, to atone, as her wrongdoings were too numerous. Occasionally she has auditory hallucinations. She refuses food; spends her time in crying; lost all interest in her surroundings.

It is a little over a year that this attack has lasted, and the patient shows no sign of improvement.

The patient's family history is interesting. Three sisters were committed twice, an uncle on her father's side is a paranoiac. At the age of ten she had epileptiform convulsions.

Such is the brief account of three typical cases of intermittent melancholia.

The evolution of the disease, as well as other features, are so

different from the common type of melancholia that they deserve a special mention.

One could see that in all of them there were no morbid symptoms immediately preceding the onset of each attack. While the ordinary melancholia develops in an insidious and progressive manner, so that the patient or his relatives cannot as a rule indicate the time when exactly the state of depression begins, in the periodic melancholia of the intermittent type the onset is strikingly sudden. Feeling perfectly well a day before, the patients awake in the morning with symptoms of sadness. Sometimes a localized physical pain will be present at the beginning, as in Case 2, in which each attack commenced with a facial neuralgia of short duration.

The duration of the attacks as well as of the intervals between the attacks presents no characteristic features, although some of the cases reported by other writers show a remarkable uniformity.

Inasmuch as the onset of the attacks is characteristic by its suddenness, their termination is equally abrupt or rapid, while an attack of ordinary melancholia disappears only gradually and very slowly.

As a last interesting observation, I wish to call attention to the state of mind of the patients between the individual attacks. It is singular to note the comparative mental lucidity, which is rather an unexpected phenomenon in view of the repetition of the attacks.

The cases which served as basis for the present study did not induce me to consider periodic insanities as a separate disease or to create a new form of melancholia. My intention was merely to present a modest contribution on an important variety of melancholia which has not received sufficient attention. I say *an important variety*, as it is not an indifferent matter for the patient and his friends to know whether he is suffering from an ordinary melancholia, which is, generally speaking, a curable mental affection, or from a variety of the same affection which has a tendency to repeat itself and become more and more aggravated with each new attack, and which by virtue of the repetition shows that there is a profound underlying degenerative make-up.

Krafft-Ebing,² from an extensive statistical study, shows how

²Allg. Zeitsch. f. Psych. Bd. 26, 1869.

great is the rôle of heredity in periodic insanities. Although melancholia belongs to the group of mental affections in which a neuropathic tendency is frequently present, in the periodic variety of melancholia a mentally morbid heredity is still stronger. This fact creates a special state of degeneration, which predisposes strongly to periodic outbreaks of insanity. Ziehen's³ careful statistical studies are in accord with Krafft-Ebing's.

The three cases which I have just reported also show the degenerative tendency.

If this is the case, the prognosis in periodic insanities is certainly extremely unfavorable; the patient may recover from an individual attack, but the tendency to a repetition will be always present. This tendency is created by a special make-up of the mentality, which cannot be radically altered. Moreover, a careful reading of the records of others and my own cases showed to me that almost each subsequent attack presented a more serious clinical picture and was of longer duration than the one preceding. It is therefore evident that periodic melancholia is of a far more serious nature than the ordinary type of melancholia.

These few thoughts suggested themselves to me as being of some importance from a practical standpoint.

³"Psychiatrie," 1894.

Society Proceedings.

NEW YORK NEUROLOGICAL SOCIETY.

April 7, 1903.

The President, Dr. Pearce Bailey, in the chair.

Birth Palsy—Syringomyelia.—Dr. Edward D. Fisher presented a man, twenty-five years of age, who had had a birth palsy, apparently the result of a tedious labor and a forceps delivery. There were present the characteristic athetoid movements, but the epileptic attacks had ceased in childhood. When fifteen years old he began to notice a wasting of the muscles of the left hand and forearm along with some disturbances of sensation. Subsequently the right hand became similarly affected. On examination, there was found to be almost complete loss of pain and temperature sense, and the tactile sense was not good. There was no sensation on either side to the strongest faradic current. There was absolute loss to both the faradic and galvanic currents in both hands. In the forearm there was a diminished reaction. The condition just described had progressed slowly during the past ten years. The reflexes were greatly exaggerated on both sides. The case had been previously diagnosed as progressive muscular atrophy, but Dr. Fisher would make a diagnosis of syringomyelia, probably originating in a slight spinal hemorrhage. The patient was very intelligent and in perfect health. He had presented no bulbar symptoms.

Dr. B. Sachs thought the association of syringomyelia with an infantile cerebral palsy was unique. The suggestion of a hemorrhage occurring simultaneously into the cervical portion of the cord at the time of the cerebral palsy appeared to him a good one. Recently cases of congenital spastic paraplegia had been reported which were known to have been produced by hemorrhage into the cord lower down. The speaker said that a few days ago he had seen a case of apoplexy in an elderly person resulting in a left-sided hemiplegia. After a few days very slight motion returned in the left hand, and he was astonished to find that he could secure fairly good motion of the paralyzed hand when asked to close the other hand. This observation was common in children, but he had not before noticed it in adults.

Dr. Pearce Bailey said that a few years ago Schultze described hemorrhages into the spinal cord occurring in young infants in connection with difficult labor, and the subject had assumed a medico-legal interest. In the cases of hematomyelia that he had seen he had never been able to observe them long enough to see the progression of the symptoms.

Suture of Popliteal Nerve; Perfect Restoration of Function.—Dr. Russel A. Hibbs presented a boy of fifteen years who had come to the Orthopedic Hospital on October 6, 1902, with a history of having fallen upon a knife seven weeks previously and injured the leg. On October 9 he was operated upon, and on exposing the site of the injury it was found that the popliteal nerve had been divided. It was accordingly sutured with a practically perfect result. While the voluntary power in the muscles supplied by this nerve was lost, there was still some sensation, which was apparently accounted for by the fact that a few fibers of the nerve remained intact. The limb was kept slightly flexed for six weeks after the operation.

Dr. M. Allen Starr thought surgeons as a rule did not recognize the great importance of suturing nerves at the time of such injuries, for, it was not uncommon for persons so afflicted to seek relief a considerable time after the injury. There could be no question that a divided nerve would not unite spontaneously in the majority of cases, and it was equally certain that by nerve suture a good result was usually secured. He had seen one or two interesting cases in which nerve suture had been essayed in children in whom the brachial plexus had been injured at birth. Four successful cases of this kind had been recently reported. The operation consisted in laying bare the brachial plexus, finding what nerves were torn, and uniting them.

Dr. William M. Leszynsky said that in injuries of the hand special care should be taken to determine whether or not the nerves had been injured. He had recently seen a case in which the divided tendon had been sutured and the divided ulnar nerve had been entirely overlooked. As a result, a neuritis of this nerve had developed before he saw the case. Although rather late, an operation had been advised in the belief that even late operations were often successful.

Dr. Fisher said that he had had under observation for some time a facial paralysis that dated from childhood, the patient being a woman of thirty. After a while a faradic response from the facial muscles was obtained; hence he thought one could hardly place a time limit after which nerve suture would afford but little prospect of benefit.

Dr. Hibbs said that in his case the original wound had been treated at a hospital, but the nerve injury had not been noticed.

Paralysis Agitans (?)—Dr. J. Ramsay Hunt presented a man of forty-three, a cigar roller by occupation. The present affection dated back four years. It had first been noticed that the right hand was weak and tremulous, but he had continued at work for three years. Last July the head also became tremulous, and the right leg felt heavy. When first seen, about one month ago, examination showed tremor of the right hand without rigidity; a slight atrophy of the right forearm; a tendency to monotony of speech and a rather expressionless face. The question arose as to whether this was an occupation-atrophy or, as he was inclined to believe, a case of paralysis agitans.

Dr. Hunt also reported a case at the Montefiore Hospital which had presented the characteristic picture of paralysis agitans for several years before death. An autopsy on the brain was alone permitted. The vessels showed marked arteriosclerosis. There was an excessive degree of pigmentation and some rounding of the processes of the cells in the cortex and in the portion of the cervical cord removed for examination—in short, those changes that had been regarded as occurring frequently in paralysis agitans. The specimens were exhibited under the microscope.

Elephantiasis Ossium.—Dr. I. Abrahamson presented a woman of thirty-six showing a peculiar glossy appearance of the skin of the forehead and a remarkably wide supraorbital ridge. Examination revealed a ptosis of the right eye, a double divergent squint, and a marked contraction of both inner fields of vision. According to her husband, the changes in her outward appearance had been progressive. The malar bones were enlarged, and apparently the hands and feet were growing larger. The condition was possibly due to luetic disease of the apophysis, because there was specific disease of the eyes.

Dr. Starr remarked that the appearance of the eyebrows pointed strongly to the case not being one of acromegaly or myxedema, but of elephantiasis ossium. The case appeared to be in an early stage.

Dr. Leszynsky said that acromegaly could be excluded by the condi-

tion of the visual field, as in that disease there was usually a bitemporal hemianopsia.

Paralysis Agitans. A General Discussion.—Dr. B. Sachs said that he was of the opinion that this was distinctly a disease of the senile period. In this period the two most important factors were emotional excitement and some prolonged and exhausting disease. The point of differential diagnosis to which Charcot had attached so much importance, the non-involvement of the head in paralysis agitans and its involvement in multiple sclerosis, seemed to him a point not well taken, for he was sure he had seen involvement of the head in fully 75 per cent of cases of paralysis agitans. Charcot's attempt to differentiate sharply between these two affections seemed to him to have been carried beyond a justifiable limit. He had seen cases giving the symptoms of multiple sclerosis, and yet later on develop a distinct picture of paralysis agitans. There was a young man at the Montefiore Hospital whom he had first seen when only about eighteen years old, and who developed the typical carriage, tremor and speech of paralysis agitans. As the years went on he developed nystagmus and a marked exaggeration of all the reflexes, and his speech was now as much that of multiple sclerosis as of paralysis agitans. He also recalled the case of a young woman first seen by Professor Mendel in Berlin, who made the diagnosis of hysterical tremor. When seen by the speaker a few months later, she presented what appeared to be the first symptoms of multiple sclerosis. At the present time, she presented the typical picture of paralysis agitans. This experience led him to think that there must be some relationship between these two diseases, and the case presented this evening by Dr. Hunt gave color to this view. With regard to the treatment of paralysis agitans he was accustomed to make use of hyoscine and the ordinary sedatives, and probably all present believed that no drug did much good. The only treatment that was fairly satisfactory was the use of some form of vibratory therapeutics.

Dr. Stuart Hart said with regard to the records of Dr. Starr's clinic at the Vanderbilt Clinic, that out of 219 cases of paralysis agitans there recorded, 139 were males and 80 females. Under thirty years of age there developed 2 cases, both males. The disease commenced in 88 cases between fifty and sixty years of age. Among heavy workers there were 45 males; among those particularly exposed to cold there were 24 cases; among the lighter trades there were 24 cases; among those working in factories were 14 cases. In 6 cases it was stated that the mother had paralysis agitans, and in 3 the father was said to be so affected, while in several other cases brothers or sisters were said to be similarly affected. Forty patients believed that the disease arose from anxiety or worry, and half of these directly attributed it to fright. In quite a number of instances the history stated that the tremor had developed very shortly after some trauma of the part first exhibiting the tremor.

Dr. Joseph Collins presented an analysis of 50 cases of paralysis agitans. Of this number 34, or 68 per cent, were males, and 16, or 32 per cent, females. The average age at which the disease developed was 51½ years, the youngest being 32 and the oldest 72 years old. They were divided according to occupation as follows: Merchants, 7; workmen, 8; laborers, 5; tailors, 4; clerks, 1; clergymen, 1; drivers, 1; carpenters, 1; plasterers, 1; engineers, 2; gardeners, 1; houseworkers, 9; captains, 1; no occupation, 7. The nationalities were: United States, 8; England, 1; Ireland, 16; Germany, 7; Holland, 1; Russia, 6; Austria, 1; not given, 10. The attributed cause was: Worry in 7 cases; alcohol, 1; excesses, 1; excitement, 1; shock, 1; refrigeration, 1; hard work, 1; pneumonia, 1; grief, 1; bite of cat, 1; unknown, 34. In 7 cases there was a history of injury; in 1 of ope-

ration; in 1 of hard work; in 1 of syphilis; while in 40 cases there was no special history of this kind. There was a history of neuropathic heredity in 13 of the cases, or 26 per cent, and a history of paralysis agitans in the parents or uncle in 6 instances. The average duration when first seen was four years, the longest being 20 years, and the shortest 5 months. The part to first show trembling was, left upper extremity in 11 cases; left lower extremity in 8; right upper extremity in 15; right lower extremity in 1; upper in 7; lower in 2; right upper and lower in 1; left upper and lower in 1; and all extremities in 4 cases. The initial symptom was: trembling in 24 cases; pain in 11; unsteadiness in 3; numbness in 2; nervousness in 1; stiffness in 1; inactivity of fingers in 1; weakness in 1; loss of power of right hand in 1; headache in 1; and not given in 4 cases. The relation of curvature of the spine was investigated specifically in only the last 20 cases, and was noted in 7 cases. He was convinced that paralysis agitans was a disease of early senility occurring as the reward of virtue.

Dr. M. Allen Starr said that he had had a fairly large experience with paralysis agitans, yet it did not bear out Dr. Collins' statement about the frequency of the disease among the Celts, and its comparative rarity among Hebrews. He could recall quite a large number of patients among Jews, and a number among the Germans. In hospital and clinical work one was apt to get a one-sided view of such matters, because of the large proportion of persons of certain nationalities at such places. Nor did his observations bear out what the last speaker had said about heredity; probably four or five per cent would represent the proportion showing hereditary influence. Anxiety and trauma appeared to him to bear a distinct relationship. He had never seen a patient absolutely cured of paralysis agitans, but he had observed a very marked improvement in one, and a great variability of symptoms in a number of other cases. The disease did not appear to be a steadily progressive one. He had found that the Swedish massage, skilfully administered, after a prolonged hot bath, gave these patients so much relief that they were willing to keep up such treatment for years. The great majority of his patients had found benefit from hyoscine in doses of 1-100 of a grain, given from two to five times a day.

Dr. Leszynsky said that dispensary and hospital patients were not benefited, but with private patients it was quite different. Four times within the past ten years he had had the opportunity of making the diagnosis very early in the disease. The disease certainly presented remissions at times, and traumatism certainly exerted a distinct influence. Most of these patients were very amenable to suggestion, and in this way could be benefited by treatment. He had not observed good effects from hyoscine except for a very short time, because most of the patients complained of the action of this drug. As systematic exercise had been recommended he had tried the Swoboda system of exercises, which call for voluntary effort entirely, and the patients appeared to be benefited thereby.

Dr. Joseph Fraenkel said that in the past ten years he had seen many cases of paralysis agitans and had witnessed 10 autopsies. It was rare to find a person suffering from paralysis agitans who was gray-haired or old-looking, and the autopsies showed a remarkable freedom from arteriosclerosis. These persons were rarely addicted to the use of alcohol or exhibited evidence of syphilitic infection, and to this extent he would indorse Dr. Collins' statement that this disease was "the reward of virtue." He saw nothing to support the theory that this was a disease of senility. It was common to observe a marked hypertrophy of the skin in these patients. A characteristic feature was the difficulty of stopping the patient when on any one line of thought and changing him to another. A pseudonystagmus was often observed. Although the tremor ceased at night, the

paralysis agitans patient presented the same attitude in sleep as when awake. He agreed with Dr. Collins that very little could be done for these patients by treatment of any kind.

Dr. Harlow Brooks said that he had only studied four cases of paralysis agitans post-mortem, and the findings were so conflicting that he was very skeptical about any lesions claimed to be characteristic of the disease. He was of the opinion that deformity of the spinal column was very common in persons who were not very well developed, basing this opinion upon many observations made in the deadhouse as well as in examining during 1898 the recruits for service in the regular army. He thought that fully 7 out of 50 such recruits would show as much spinal deformity as Dr. Collins' cases, even though these persons were of sufficiently good physique to be accepted in the army.

Dr. Collins said that his statistics with regard to the Celts might be refuted, but to do so the actual figures must be presented and not cases recalled from memory. He believed also that the more paralysis agitans was treated the worse it became.

Dr. Hunt said he understood that the premature senility referred to the nervous system, and not to the arterial tree when used in connection with paralysis agitans.

Dr. Pearce Bailey was of the opinion that two-thirds of the cases of paralysis agitans began after the age of sixty. A disease which could become fairly well developed in the course of a few weeks could hardly be regarded as an organic disease of the nervous system, and he was inclined to think that the evidence was rather in favor of its being a disease of the muscular system, resulting possibly from the action of certain toxins. He believed it was distinctly induced by trauma, either psychic or physical, for there were too many definite and striking clinical records of this kind. The disease usually first appeared in the hands, and yet in cases of injury to the legs the disease was more apt to begin in the legs. A large proportion of the persons coming to Dr. Starr's clinic suffering from paralysis agitans were apparently right-handed workers.

A Specimen of Brain Abscess.—Dr. Joseph Collins presented this specimen removed from an Italian laborer, forty years old, who had not been sick up to two months before his admission to the City Hospital, since which time he had suffered from headache only. On January 14 he became stuporous. There was no paralysis, paresis or spasm, in fact, no objective symptom other than stupor. Examination on February 3 showed the head to be drawn over to the right side and flexed, and the right upper extremity partly flexed at both joints. There was a peculiar elastic resistance of the right upper extremity on extending it. Knee-jerks were absent. The plantar jerk on the right side was lively, but could not be elicited on the left side. The body temperature remained about normal, and the pulse varied from 68 to 90, and the respirations between 20 and 22. Dr. Collins' diagnosis was circumscribed non-purulent encephalitis, and because of the motor symptoms on the right side the lesion was thought to be in the left cerebral hemisphere. However, the lesion was found to be a focus of comparatively recent yellow softening situated on the right convexity, involving that portion of the first temporal convolution lying immediately below the supramarginal and angular gyri, and separated from these by the Sylvian fissure. It involved the posterior arm of the internal capsule, and extended apparently into the extraventricular radiations emanating from the lower third of the posterior central gyrus. The internal capsule was not involved in its passage between the basal ganglia. No thrombus could be found. The special interest of the case lay in the fact that the motor symptoms were on the right side and the lesion was found on the same side.

Periscope.

MISCELLANY

Cardiac Neuroses.—Pick, in his capacity as the chief of the Vienna Military Hospital, observed 41 soldiers between eighteen and thirty years of age, with a syndrome of symptoms approaching the character of Basedow's disease, but at the same time differing so essentially from it as to merit special consideration. The patients suffer from palpitation and oppression at the least effort, and complain of muscular fatigue. At the physical examination they all presented symptoms of a certain degree of a hypertrophy of the thyroid gland, usually of the left lobe, but the goiter was never pulsatile. The pupils are unequal, the right one rather dilated, but reacting to light; the mydriasis can hardly be ascribed to pressure of the right sympathetic through the lobe of the thyroid for the hypertrophy of the latter is too insignificant to cause such disturbance. All the subjects suffer from a very irritable heart, and the slightest effort suffices to increase abnormally the heart beats; so that in one minute the beats will jump from 70 to 140 per minute. The heart, as asserted by Kraus in other cases of thyroid hypertrophy, is rather dilated than hypertrophied. The cutaneous capillaries are very irritable and all the patients present marked dermatography. Axillary hyperidrosis, anesthetic pharynx, but without other symptoms of hysteria. Some show tremor of hands when extended with eyes closed, others some cardiac arrhythmia. Unable to form a positive diagnosis the author supposes the syndrome of symptoms to be a disguised form of Basedow's.

JELLIFFE.

The Various Reflexes in Diabetes.—It has been observed by Landouzy Rosenstein and others that the patellar reflex is either diminished or entirely absent in cases of diabetes mellitus. Pitres (*Soc. de Bul. de Bordeaux*, Nov. 11, 1902) undertook to investigate the condition of other reflexes in this affection. With this in view he examined 32 diabetics, and he finds the following:

	<i>Patellar.</i>	<i>Cutaneous.</i>	<i>Cremaster.</i>	<i>Plantar.</i>	<i>Pupillary.</i>
Abolished	13	6	19	16	1
Weakened	7	8	6	2	0
Exaggerated	2	6	4	6	0
Normal	10	1	3	8	31

It would thus seem that the cutaneous reflexes are, even more often than the patellar, either abolished or weakened. At times the tendon reflexes are first to disappear, at other times it is the cutaneous; of the latter we find at one time the cremasteric weakened or abolished, at other times the abdominal or the plantar, while the others are either exaggerated or normal. This great variability is observed also in other pathological states, especially in tabes, and this renders it somewhat difficult to explain the significance of the loss of reflexes. Contrary to the above, the pupillary reflexes are almost always preserved in diabetes. In the single case in which the author found them absent there was arteriosclerosis, diabetes with glycosuria and moderate polyuria. In the other cases the reaction of the pupils to light, accommodation and pain were perfectly normal. The conclusion may thus be drawn that although the condition of the cutaneous and tendon reflexes may not seem in the way of a differential

diagnosis between true tabes and diabetic pseudotabes, the character of the pupillary reflex and of the epigastric sensibility as well as the cremasteric reflex may be utilized in difficult cases to distinguish one affection from the other; inasmuch as the Argyll-Robertson pupil and the epigastric and testicular analgesia, so common in true tabes, are not observed in diabetes.

† ELLIFFE.

RIVISTA SPERIMENTALE DI FREMATRIA

(Vol. xxviii, Fasc. iv, Dec., 1902.)

1. The Correlation between Psychic and Organic States. SILVIO TONNINI.
2. A New Anomalous Process in the Sphenoid Bone of Man. TENCHINI and ZIMMERL.
3. Peripheral Facial Diplegia. PANEGROSSI.
4. A Contribution to the Study of Precocious Malignant Syphilis of the Brain. PANICHI.
5. The Toxic Principles of *Aspergillus Fumigatus* and *Flavescens* and Their Relation to Pellagra. CENI and BESTA.
6. The Nervous Lesions and the Pathogenesis of Amyotrophies of Articular Origin. PIGHNI.
7. A Contribution to the Study of Lingual Hemiatrophy. BIANCONE.
8. The Toxicity of the Blood of the Insane. CENI and PINI.
9. Hemorrhagic Encephalitis with Special Reference to the Tuberculous Variety. BOMBICCI.
10. Hypothermy in the Epileptic. BESTA.
11. The Assistance Rendered to Insane in Italy. TAMBURCNI.
12. The Action of Gastro-intestinal Juice on *Aspergillus*. CENI.

1. *The Correlation between Psychic and Organic States*.—The author employs the term correlation to express that close and varied connection which unites and co-ordinates the organic functions with the psychic ones, and these last with the psychic acts as manifested to the eye, etc. Disease, degeneration, mental sanity have all their proper psychic and psycho-organic correlations, and these correlaries are to be found not solely in pathologic conditions; for correlation is a biological function, and without it there is no life. To give an idea of the importance practically of these organic and psychic correlations, it is sufficient to take some examples from the sphere of the sexual life which exerts such a direct influence on the development and orientation of psychic life. Thus sexual perversions are always associated with certain organic and psychic secondary sexual characteristics. The author brings forth a case in point, that of a man of fifty-seven who was accused of seducing a young woman, but who claimed to be impotent. Physical as well as a thorough psychological examination by the author concerning all the man's habits, mode of thinking, etc., proved to the jury conclusively that the man could not but have been perfectly potent. The author makes the proposition that psychological examination and investigation should be carried out not only in cases of suspected mental alienation, but in all cases that lack proof of a committed crime; as such an examination requires knowledge and skill it should generally be entrusted only to an experienced psychiatrist. This province has, however, been invaded to some extent by Criminal Anthropology, with its practical tendencies to fix the correlation between the physical and psychical character of certain criminal types. Thus the mobile physiognomy, the erratic look, the uncertain gait of the thief are but dynamic symbols approaching the author's psycho-organic correlations. A great importance is to be attached to gesture, to various signs and motor phenomena which very frequently serve as substitutes for language. The author brings forth examples

from the creations of the great writers of the present time (as Tolstoi, Victor Hugo, Zola, Bourget, etc.), showing how their deep psychological insight into human nature enabled them to comprehend and present in their works the correlation between the physical and psychic states of the presented types.

2. *A New Anomalous Process of the Sphenoid Bone in Man.*—Of interest to the student of anatomy.

3. *Peripheral Facial Diplegia.*—Double paralysis of the facial nerve of peripheral origin, although known to Christison (1835), Bell (1836), and others, is of rather rare occurrence, and quite difficult to diagnose; nor is the etiology of the affection always easy to determine. The author describes three cases with an undoubted clinical picture of the affection. What strikes the observer in all the three cases is the fixed look and the peculiar aspect of the immobile, expressionless, mask-like face, the perfectly smooth forehead, the eyes open wider than normal, the nostrils fallen in, the naso-labial sulcus smooth, the cheeks flattened, the lower lip hanging down. The patients were unable either to frown, or to close their eyes; could neither spit, whistle nor blow, and also found it difficult to pronounce certain letters—all this showing that the motility of all the muscles of the face that serve mimicry was completely abolished. In differentiating this paralysis from a central nuclear affection of the nerve the author considers first the fact that both the inferior as well as the superior facial were involved in the paralysis; secondly in one of the cases the paralysis was localized exclusively in the area occupied by the seventh nerve, an occurrence which is frequent in paralysis of peripheral origin, but rather rare in that of nuclear affection, in which together with the facial there are also attacked other cerebral or spinal nerves. As distinguished from a labio-glosso-pharyngeal paralysis it is to be noted that this last, with very few exceptions, does not extend to the upper part of the face, nor is mimicry in any way affected, and while in peripheral facial diplegia both sides of the face may be attacked simultaneously, in the labio-glosso-pharyngeal paralysis the lips suffer first, and then progressively the tongue, the palate, the larynx, etc., muscular atrophy ensues alongside of the paralysis and both advance slowly. The slowness in the advance of the disease, and the early atrophy as found in superior poliomyelitis serve to exclude this malady; moreover, in this affection the paralysis of the facial muscles is always associated with that of the muscles of the eye, a rather rare occurrence in facial diplegia. Atrophic paralysis of the muscles of the face is also observed in certain cases of progressive muscular atrophy of the juvenile type of Landouzy-Déjerine. But the whole facial aspect of these cases is different from that presented by the patients in question, and this facial appearance is but an insignificant part of the clinical picture of the affection; moreover, in these cases the atrophy and paralysis of the muscles of the face is even of a slower development than in labio-glosso-pharyngeal paralysis. From the time of Christison (1835) up to Davanil (1850) there was but one monograph in the literature of this affection; next we find Wachsmuth (1864), who was the first to understand the disease better than his predecessors and who gave to it the name of diplegia. He was followed by Pierreson in 1867, and then of late Stintzig (1893), Hirbschman, Bregman, and quite recently Raymond, have all devoted considerable time to this disease. The author presents an elaborate and very instructive summary of ninety cases of this affection, so far found in literature, with the designation of the symptoms, course, etiology and termination of each. This summary is valuable enough to present by itself a full monograph on the subject.

4. *Malignant Syphilis of the Brain.*—The patient, a man of twenty-

seven years, was admitted to the hospital with paresis and a beginning disturbance in the motility of the tongue. This condition seems to have ensued rapidly during one night; and to it were soon added paralysis of the left half of the face, conjugate deviation of the eyes to the right; continuous yawning. The anamnesis of the patient is somewhat indefinite, except that he became recently a hard drinker, and contracted syphilis over a year previous to admission; two months previous to this admission he began to suffer with irregular fever, and vague pains in the articulations; more recently with intense headache, of a diffuse and continuous character. During observation in the hospital the cardiac symptoms (formerly mitral insufficiency) grew worse, and the frequency of the heart beats increased up to 130 and 160 per minute. Notwithstanding the energetic anti-syphilitic treatment resorted to while in the hospital the improvement was very insignificant. A month before the fatal issue the condition of the patient was as follows: Patient prefers to lie on right side with head and eyes turned toward same side; when grinding his teeth the left corner of mouth remains immovable; in frowning the left side of forehead is almost flat; eyes present some nystagmus. The tongue is deviated toward the left, but is quite mobile; shows some slight fibrillation. The left upper extremity is flexed at a direct angle and lies near the trunk, but preserves voluntary as well as passive motions. The left lower extremity is bent at an obtuse angle to the knee, admits of no movement whether active or passive. Patient urinates freely, but the function of defecation is somewhat affected. Left pupil wider and less active than the right. All the reflexes abolished on the left side; thermic and tactile sensibility considerably diminished on same side; hearing also abolished on same. Bilateral hemianopsia of the right halves of both retinae. The patient remained almost all the time in a state of sopor, from which he would awake with some difficulty. The anatomical diagnosis on autopsy was as follows: A dissecting aneurism of the second branch of the right Sylvian artery (middle cerebral); aneurism of the fourth branch of the left Sylvian; softening of the gyri of dextra insula, and hemorrhage in the right capsule of the thalamus. The microscopic examination of the lesions established beyond doubt the syphilitic origin of the disease (syphilitic arteritis, syphilitic aneurism).

5. *The Toxic Principle of Aspergillus*.—An experimental study on the toxicity of *Aspergillus fumigatus*.

6. *Amyotrophies of Articular Origin*.—A continued article.

7. *Hemiatrophy of the Tongue*.—A continued article.

8. *The Toxicity of the Blood of the Insane*.—A rather unsatisfactory account of a series of experimental studies to determine the toxicity of the blood of patients suffering from various forms of insanity. The authors are forced to confess that notwithstanding the considerable number of experiments it was impossible to establish definitely any difference between the serum of normal individuals and that of insane. Whatever toxicity was demonstrated was observed in the blood of maniacs and epileptics. The toxic power of the blood of individuals affected by various psychopathic maladies (maniacs, alcoholics, paralytics, paranoiacs, epileptics, etc., did not present any notable variations; nor was there any variation in the different forms of one and the same disease, as in the periods of calm and excitement. It would seem as if the present methods of experimentation were not sufficiently reliable to enable one to control and definitely establish the degree of toxicity of the blood serum in the various maladies in question.

9. *Hemorrhagic Encephalitis*.—A continued article.

10. *Hypothermy in Epileptics*.—The case serving as the basis or this

observation was that of an epileptic, twenty-eight years of age, who had been suffering from epilepsy for the last ten years. During the last two years the attacks occurred four or five times a month and were followed by a state of depression and mental confusion. Patient is rather of a taciturn disposition, but well physically and without any degenerative stigmata. Nutrition good, functions of organic life normal. The morning temperature was 35.2 deg. and kept low for about 18 days; during this time there occurred oscillations between 35.1 deg. and 36.4 deg. There was no symptom to indicate any abnormal condition or even slight disturbance whether physical or mental. The author ascribes this low temperature to some special condition of the metabolic processes of the epileptic. It is quite possible that the same toxic agent which acts on the nervous system producing the condition of epilepsy may be the cause of the hypothermy in these cases.

11. *The Condition of the Insane in Italy.*—An historical essay on the present condition of the asylums for the insane in Italy, showing a recent increase in the number of the insane population of the land, and an overcrowding of the institutions for the maintenance of the insane. The author proposes various provisions for relieving the congestion in the asylums, and especially a system of family care of the insane.

12. *The Action of the Gastro-Intestinal Juice on the Spores of Aspergillus.*—This is a complement to article 5, on the relation of the spores to the genesis of pellagra.

ALEX. ROVINSKY (New York).

NEUROLOGISCHES CENTRALBLATT

(1903, No. 3, Feb. 1.)

1. The Isthmostriatric Tract (or Bulbo-striatic Tract) of Pigeons. A. WALLENBERG.
2. A Remarkable Complication of a Case of Migraine. L. HOEFLMAYR.
3. A Contribution to the Knowledge of Disseminated Encephalomyelitis. E. BANCKE.
4. Ear Reflex. W. ALTER.

1. *Isthmostriatric Tract.*—Wallenberg failed to obtain degeneration of the isthmostriatric tract in a pigeon by unilateral disturbance of the cerebellum even if the central nuclei of the cerebellum were involved. Division of the sensory root of the fifth nerve between the bulb and the Gasserian ganglion resulted in degeneration of the dorsal and lateral periphery of the nuclei. It was then possible by the Marchi method, to trace the tract as far as the anterior portion of the base of the brain. In the frontal lobe this tract of fibers divides into four bundles, which terminate in different parts. The terminal sensory trigeminal nucleus, therefore, is in direct union with the basis of the frontal lobe without the intermediary of the thalamus.

2. *Migraine.*—A woman, fifty-seven years of age, who had ceased to menstruate six months before. Father and sister had suffered from migraine; one of her children was hysterical and had headache. Patient had migraine for forty years. Lately attacks were more frequent and constipation and the menstrual flow would aggravate symptoms. Had taken large doses of migrarine every day previous to special attack. This attack began with a pulling up of one eye and one eyeball, hemiatropia, collapse and the fear of death. She was semiconscious for several days without sleeping. When disturbed would repeat the syllables "noten" and "nieten." There was obstinate constipation and agrypnia. On the sixth day the face assumed an expression of pain. Twitchings of both arms and legs appeared. During sleep she was quiet. On the tenth day consciousness returned, after

which she improved slowly, but had occasional hallucinations and some disturbance of the field of vision. The symptoms were those of ophthalmic migraine and toxine infection. It is possible that coprostasis was the exciting cause.

3. *Disseminated encephalomyelitis*.—To be continued.

4. *Ear Reflex*.—Alter describes a new skin reflex which is obtained by striking the ascending ramus of the inferior maxilla resulting in a vigorous contraction of the attoleus aurem with elevation of the ear. He found this in a parietic dement with left-sided facial paralysis and status epilepticus, by striking on the right side. On investigation he found it 19.4 per cent of patients with parietic dementia. It differs considerably on the two sides. Alter does not arrive at a definite conclusion regarding its meaning.

(1903, No. 4, February 15.)

1. A Contribution to the Casuistry of the Relation of Hair in Cases of Mental Diseases. W. HEINICKE.
2. The Biology and the Functional Activity of the Central Nerve Cells. P. KRONTHAL.
3. A Contribution to the Knowledge of Disseminated Encephalomyelitis. E. BANCKE.

1. *Hair in Mental Disease*.—Henicke reports the case of a girl twenty-one years of age suffering from dementia præcox. Symptoms had first appeared at the age of thirteen. She had occasional periods of excitement, in one of which it was observed that the pupils were dilated ad maximum and that an almost silver white band of hair passed from the forehead to the right parietal bone. The hair remained in this condition for four days and then rapidly returned to its normal condition. Three days later a second attack occurred, accompanied by a similar dilatation of the pupils, but the coloration of the same band of hair was observed one to two hours before attack. During the height of the attack the hair became almost silver white. The following day it returned slowly to its normal blonde color. Microscopical examination showed that this change of color was due to the filling up of the sheath with air, and when the air was expelled, the hair returned to its normal color. The manner in which the air accumulates cannot be determined.

2. *The Central Nerve Cells*.—Kronthal begins with a consideration of phenomena which follow stimulation of the gray matter of the cortex. These he considers as due to the excitation of the nerve fibers. It is noteworthy that changes of diet do not affect the nerve cell, and that the central nervous system, even in starvation, suffers less than any other part of the body. Some substances, as narcotics, act upon nerve cells as upon other forms of protoplasm. Ganglion cells in the process of division have never been found. The hypothesis that man is born with a large mass of reserve cells he assumes as impossible. Kronthal occasionally has observed a strange cell in the nerve cell which he thinks is a leukocyte. Kronthal believes, therefore, that the nerve cell is due to a coalescence of leukocytes, that it is no organism, and that it is not alive. The functions of the nervous system are accomplished by the neurofibrils which unite the nerve fibers. The functions of the nerve cells have not been definitely determined.

3. *Disseminated Encephalomyelitis*.—A school teacher, thirty-nine years of age, with mental symptoms of dementia præcox, had meningitis in childhood. Five and one-half years before death she began to limp on left side. Examination showed excessive spasticity and contractures in left leg, the latter disappearing completely during chloroform narcosis. A plaster cast was put on, which, after 17 days, had to be removed on account of extensive decubitus. About a month later paralysis of the bladder and

rectum and lower extremities, with loss of sensation, appeared. Later still paralysis of the right arm. After four months she died. There was a fresh internal hemorrhagic pachymeningitis. Microscopically numerous areas of degeneration in various stages were found throughout the brain and cord, which are described in detail. A secondary gliosis was present which was characterized by the presence of large cells with oval nuclei. Bancke believes that the case was one of dementia præcox in which infection was produced by the bed sore. T. H. WEISENBURG (Philadelphia).

MONATSSCHRIFT F. PSYCHIATRIE UND NEUROLOGIE
(1903, April.)

1. A Case of Isolated Agraphia. C. WERNICKE.
2. Hyperesthesia of the Nails (Onychalgia nervosa). H. OPPENHEIM.
3. Epileptic Mania with Remarks upon the Loss of Ideas. H. HEILBRONNER.
4. The Origin and Course of the Oculo-Motor Nerve in the Mid-Brain. N. MAJANO.

1. *Isolated Agraphia*.—A woman forty-six years of age noticed a gradually increasing weakness of the right hand which suddenly increased to paralysis of the right arm and leg with contractures. Eight days later there were twisting of the mouth, and complete loss of speech, although the patient appeared to understand what was said to her. Gradually speech improved and then grew gradually worse and remained stationary. At this time there was paralysis with spasticity of the arm and leg, anarthria, good repetition, and some spontaneous speech. She understood words fairly well, but there was complete agraphia. Memory was defective and the patient remained in this condition during all the period of observation. Therefore the predominant symptom and the only complete one is the isolated agraphia. Only a few similar cases are reported in the literature. The characteristic of all these cases is that the lesion is on the left side. Nevertheless, Wernicke does not share the opinion of Dejerine that there is a unilateral optic word center, at least he does not believe that the existence of such a center is proved by these cases.

2. *Hyperesthesia of the Nails*.—Oppenheim reports three cases characterized by extraordinary sensibility of the finger-nails so that even slight mechanical injuries caused intense pain. Even trimming the nails caused pain. In all cases there was congenital neurasthenia. There are a few references to this condition in the literature, although it does not appear to have been recognized as a particular form of hyperesthesia.

3. *Epileptic Mania*.—Heilbronner continues his article with a discussion of his cases. He calls attention to the undoubted false perception that sometimes occurs in epileptics, and then commences to discuss the flow of ideas. He does not believe that there is any necessary relation between these and the readiness of speech, in fact in many cases speech may be distinctly inhibited although the patients complain of a continual series of thoughts passing through their heads; that is to say, there may be a flow of ideas without any motor excitement. It does not appear that the flow of ideas is the result merely of an increased psychological motor irritation, not even in mania. In this condition there is motor excitement, but the flow of ideas is independent of it. It is always important to compare the inclination to speech with the psychical representations that the patient has prepared. It must be admitted, however, that the whole subject is in a very unsatisfactory state.

4. *Oculomotor Nerve*.—Majano continues his article with a careful study of the literature of the origin and course of the oculomotor nerve.

He concludes partially as a result of these studies, and chiefly from his own experience, that the sublongitudinal or predorsal fasciculus has its origin in the anterior nucleus of the anterior corpora quadrigemina; that the ventral portion of Meynert's ligament arises from the dorsal median region of the corpora quadrigemina; that the sublongitudinal fasciculus passes obliquely forward and downward toward the median line, enters the capsule of the red nucleus, and some of the fibers then pass down to the root fibers of the oculomotor, but the main portion decussates and unites with the root fibers of the oculomotor of the opposite side. The mechanism of the pupillary reflex then would consist of the following three parts: the peripheral sensory neurone arising from the bipolar cells of the retina and forming the pupillary fibers of the optic nerve which pass to the dendrites of the ganglion cells of the lateral portion of the anterior corpora quadrigemina. Second, the central neurone which consists of the ganglion cells of the lateral portion of the anterior corpora quadrigemina, and the direct or crossed fibers of the predorsal fasciculus which unite with the root fibers of the oculomotor nerve, and with the collaterals of the axiscylinder and sympathetic cells of the ciliary ganglion of the same or the opposite side; third, the peripheral motor neurone, which consists of the cells of the ciliary ganglion and the fibers arising from them which innervate the sphincter of the iris. The pupillary reflex is, therefore, primarily dependent upon the anterior corpora quadrigemina of the opposite side. The direct pupillary reaction is stronger than the indirect on account of the double decussation of the neurones concerned in it. The unilateral reflex paralysis is more pronounced on one side, occurring when the neurones between the anterior corpora quadrigemina and the ciliary ganglion are degenerated. If the ciliary ganglion and the ciliary nerves are destroyed then total paralysis occurs. The consensual reaction is not destroyed by longitudinal section of the chiasm.

J. SAILER (Philadelphia).

DEUTSCHE ZEITSCHRIFT FÜR NERVENHEILKUNDE

(Band 22, Heft 3-4.)

12. Skin Reflexes and Their Nerve Tracts. MUNCH-PETERSEN.
13. Contribution to the Knowledge of Tumors of the Spinal Cord. MEYER.
14. Multiple Sclerosis Developing into a Picture of Transverse Myelitis. FLATAU and KOELICHEN.
15. Acute Cerebral and Cerebrospinal Ataxia. LÜTHJE.
16. Contribution to the Diagnosis of Tumors in the Frontal Lobe. AUERBACH.
17. A Case of Relapsing Bilateral Ptosis, with Myasthenic Manifestations in the Upper Extremities. BYCHOWSKY.
18. Cystic Meningitis in Racemose Cysts of the Central Nervous System. ROSENBLATH.

12. *Skin Reflexes and Nerve Tracts.*—Munch-Petersen contributes a valuable article upon the reflexes of the skin, first discussing the history of some of the more important reflexes, and then defining them as involuntary movements which are produced by irritation of the skin, he then speaks of the atypical form which can be obtained in almost any part of the body, and then mentions the so-called typical forms which include the plantar, cremaster and abdominal reflexes. The degree of irritation required indicates inversely the activity of the reflex. There seems to be some relation between the skin reflexes and the tickling reflexes. He believes the plantar reflex has a single reflex center; discusses most thoroughly the Babinski phenomenon, calls attention to the fact that there is dorsal flexion of the toes during walking in persons who possess it; and discusses some

interesting time estimations that he made upon this reflex by the aid of kinometographic studies. Investigations have convinced him that the movements of the toes are irregular when the soles of the feet are irritated. He believes that there is a distinct relation between the gait and the plantar reflex, that is to say, they are typical or atypical together. With regard to the arcs by which these reflexes are accomplished, he makes the following statements. When sensation is lost the reflex is lost; when increased the reflex is increased; therefore, sensation is an essential condition for the reflexes. If, however, the reflex is produced by sensation, the sensitive part of the reflex arc must have its center in the cortex of the cerebrum. The relation of the plantar reflex to the gait indicates that it has some relation to a motor center in the Rolandic cortex. Analogy indicates that the skin reflexes have their sensitive as well as their motor centers in the cortex of the cerebrum. A study of these reflexes in different conditions seems to lead to the same result. In diseases of the peripheral nerves or in total lesion of the spinal cord the skin and tendon reflexes were entirely lost. Voluntary motion and sensation were also lost, and, therefore, there was reason to believe that the tracts of these different functions were identical as far as the point of solution of continuity. Munch-Petersen has found that when the plantar reflexes were in part preserved the motor tracts were not entirely destroyed. Studies of apoplexy also lead to the conclusion that at least as far as the internal capsule, the reflex tracts and the tracts for voluntary motion were identical. In cases of recent apoplexy with a persistent plantar reflex the prognosis is more favorable. Further, in those cases in which there is ataxia in motion, dorsal flexion during walking is found in those cases in which the Babinski phenomenon is present. Babinski's phenomenon he believes is due to a degeneration of the pyramidal tracts which gives rise to a paralysis of the plantar flexures or to ataxia. In hysteria the skin reflexes are influenced, but show no qualitative alterations from the normal. During hypnosis suggested anesthesia diminishes or abolishes the skin reflexes; suggested hyperalgesia increases them. In conclusion Munch-Petersen calls attention to the following experiment. If the sole of the foot is irritated with a needle the foot is drawn upward; if a threat is made to irritate the sole of the foot with a needle, the foot is also drawn up, finally in some cases the mere sight of the needle is sufficient to produce an involuntary movement of the foot. Therefore, we cannot state certainly that the skin reflexes are not produced by an act of the will, but only that this activity of the will does not reach the highest phase of consciousness. The skin reflexes are in all probability located in the cortex of the brain.

13. *Spinal Cord Tumor*.—A girl fourteen years old had severe pain and paresthesia of the left upper extremity accompanied by some weakness. Fifteen months later she developed spastic paralysis of the left leg; three months later there was flaccid paresis of the right arm and spastic paresis of the right leg. About the same time she had pain in the middle of the cervical region of the spinal column and tenderness upon pressure over the fourth and fifth vertebrae. In the course of the next six months she developed almost complete spastic paralysis of the legs. There was some atrophy of the arms on both sides and hypalgesia and hypesthesia on the ulnar sides. There was some disturbance of the bladder and rectum. The order in which the symptoms appeared was rather against tumor of the spinal column, and they were rather difficult to make agree with tumor of the cord itself, therefore there was reason to believe that the meninges were involved. It was not possible to decide whether it was extra- or intradural although the early development of the bone symptoms speaks in favor

of the former. These symptoms Meyer believes in agreement with Bruner, are not trustworthy. The segments or roots involved were apparently the fifth, sixth and seventh, and probably on account of the hypesthesia and hypalgesia on the ulnar sides of the arms, the eight cervical and first dorsal segments. The clinical symptoms were remarkable, partly for the variation from time to time in the areas of disturbed sensation, and the frequent involuntary flexion of the legs. At the autopsy the stomach was found to be enormously dilated. This was possibly the result of the lesion of the spinal cord. A tumor was also found on both sides of the dura, constricted at the point at which it passed through this membrane. It was about three by one cm. in size, shaped something like an egg; its surface was smooth, it was firm in consistency, and directly involved the sixth, seventh and eighth cervical and dorsal segments. It also involved the left side of the cord and pressed from the anterior surface backward. Histologically it proved to be a fibro-sarcoma. Operation was not performed partly because hypertrophic cervical pachymeningitis was suspected, and partly because the condition of the patient was so bad.

14. *Multiple Sclerosis and Transverse Myelitis*.—Flatau and Koelichen report the case of a woman sixty years of age who had been exposed to cold. After this she developed fever, had pain in the legs, no control of the bladder, and obstinate constipation. She became bedridden, was unable to walk or turn over in bed; there was almost complete paralysis of the right leg, although she was able to flex the thigh, and still had slight power of adduction; there was almost complete paralysis of the left leg, the movements being even less than on the other side. There was slight spasticity of the flexors of the right thigh. Although the legs were emaciated there were no muscular atrophies. There were some slight sensory disturbances, loss of the patellar reflex on the left side and great diminution on the right. Severe irritation of the sole of the foot caused withdrawal of the leg. There was a bedsore over the sacrum and over both trochanters. The patient gradually grew worse and died. At the autopsy nothing was found macroscopically in the brain and spinal cord. A very careful description with illustrations is given of the results of the Kneisel, Weigert and Marchi methods. The changes found could be classified in two groups: first, early degeneration with degeneration of the myelin fibers not involving the blood vessels or neuroglia; and second, advanced areas of degeneration with involvement of the blood vessels and the neuroglia, and the presence of a great number of compound granular cells. The authors regard the case as an atypical form of multiple sclerosis. There were very slight changes in the nerve cells and in the axis cylinders, which they consider characteristic of this disease, and they classify multiple sclerosis therefore as a diffuse process involving inflammation of the blood vessels of the nervous system, especially the spinal column. In an editorial note Strümpell states that he believes the case to be one of acute disseminated myelitis, and not multiple sclerosis.

15. *Cerebral and Cerebrospinal Ataxia*.—Lüthje reports the following three cases of acute ataxia. A boy of ten years was suddenly attacked by headache, prostration and vomiting. There were no mental symptoms for the first two days, when the patient became violently delirious, there were signs of bronchitis in both lungs with a roseola eruption. The fever lasted for about five weeks, gradually falling to normal. The patient throughout the greater portion of the time was unconscious, then became confused, had to be fed, had peculiar twitchings in the muscles of the face. The Widal reaction was positive. A month after the temperature had become normal the patient began to show signs of returning intelligence, and a

month later it was impossible to make a careful examination. At this time, aside from a rather exaggerated emotional state, his intelligence was normal. Speech was deep, somewhat nasal and monotonous, pauses frequently occurred between words and syllables, and the different syllables were sometimes enunciated in an explosive manner. The motor power was not altered. At any attempt at voluntary motion, however, there was extreme ataxia and sometimes associated movements of the other arm when one of the upper extremities was moved. Walking was entirely impossible; sensation was normal; the reflexes were increased; and there was ankle-clonus. The patient gradually improved, was able to sit up alone, the ataxia disappeared to a large extent, although it was more pronounced in the left than in the right arm, and was still evident in the legs. There was bilateral disturbance of the stereognostic sense. Two months after convalescence from the fever, speech was still gravely disturbed. The second case, a sister of the first, nine years old, was brought to the hospital suffering from a severe attack of typhoid fever. She was unconscious for weeks, and five weeks after onset of the disease began to have twitchings in the face and rhythmical lateral movements of the head. During convalescence examination showed that speech was toneless, the syllables were pronounced slowly, occasionally explosively; there was marked ataxia in the movements of the arms and legs, especially the latter; the patellar reflexes were exaggerated, and there was distinct ankle-clonus. When the patient attempted to stand there were violent oscillations which were not increased by closing the eyes. Sensation was not disturbed. She gradually improved, but when discharged had difficulty in walking, and the speech was still monotonous and scanning. The third case, a six-year-old brother of the two preceding cases, was brought to the hospital suffering from typhoid fever, which also proved to be a severe attack. During the attack he had dilatation of the right pupil, was unconscious, later confused, and also developed rhythmical lateral movements of the head. When excited he had disseminated erythematous eruption over the head and face, exquisite ataxia in the head and trunk, and no control of the sphincters. There was no disturbance of sensation, but the stereognostic sense was not tested. Speech was slow and monotonous and the syllables were separated. The tendon and skin reflexes were exaggerated. This patient rapidly improved and was discharged almost well. The cases are very similar, and Lüthje believes they belong to the pure cerebral form of acute ataxia. In one case, however, the symptoms indicated involvement of the cerebellum. He discusses the differential diagnosis of spinal, bulbar and cortical ataxia and is inclined not to exclude the latter because hemiparesis and hemiplegia did not exist; because the clinical course showed that whatever the lesions were they were exceedingly slight. He believes that the disturbance of the stereognostic sense is one of the most important signs of the cerebral origin of the disease. The disturbance of speech did not indicate bulbar, but rather cortical involvement. He concludes with a careful study of the literature, having found and abstracted sixteen cases, in many respects similar to his own. Some of these were purely cerebral, and a few evidently cerebrospinal in origin. In an annotation he states that four months after the patients were discharged the two younger were almost entirely well; speech was slightly slow, and the patellar reflexes were exaggerated. In the first case the speech was still scanning and monotonous and the reflexes were exaggerated and there was still slight ataxia in the extremities.

16. *Frontal Lobe Tumors*.—Auerbach reports the case of a woman forty-eight years of age who had always suffered from migraine. Usually

she had been industrious, but she began to neglect her work, became irritable, complained of fatigue, had increasing headache, and often refused to leave her bed. From time to time she vomited. The condition gradually grew worse, she ceased to take interest in things, often refusing to eat unless urged. She was apathetic, unclean, and slept considerably. There was some swelling of the face. The nervous system, with the exception of increased patellar reflexes without ankle-clonus or Babinski's phenomenon, and slight tremor of the hands on motion, was negative. Intelligence seemed to be considerably diminished. Memory for recent events was impaired; there was no tendency to joke. There was a suspicion of myxedema; the patient was given thyroid tablets without benefit. She had an apoplectiform attack during which she was unconscious for half an hour, but subsequently recovered, when it was noted that she had difficulty in fixing the eyes, the pupils were dilated, swallowing was difficult and the patient was bent forward and complained of pain in the spinal column. Examination of the eyes showed choked disks. Later she developed stiffness of the neck, and pain upon movement of the head, gradually grew worse, became comatose and died. At the autopsy the frontal lobes were found flattened, the dura was adherent in this region, and the veins were more congested here, especially over the left frontal lobes. The base of both frontal lobes was occupied by a tumor about the size of an apple, extending from the anterior perforated substance forward. The surface of the tumor was somewhat nodular; the mass had about the consistency of liver tissue, and the color varied from pink to dark red. Microscopically it proved to be a fibrosarcoma, probably arising from the dura of the base. The diagnosis presented many difficulties. At first Professor Edinger, who also saw the case, thought of precocious senile dementia, or arteriosclerosis in the vessels of the brain. Later tumor of the frontal lobes was suspected, but the only symptoms were the headache and the psychical disturbances. Of the latter, disturbance of memory and loss of the power of attention, the loss of any initiation or apparently voluntary action, were the most prominent features. Auerbach discusses the theory that intelligence is located in the frontal lobes. He believes that in the human brain they are to be regarded as the most important center of association. Among the interesting symptoms are the apoplectiform attack, the occurrence of choked disks, the absence of disturbance of equilibrium, and the absence of a tendency to make jokes. There is apparently no other case in which the tremor of the hands was so marked. Among the negative symptoms it may be mentioned that the temperature was not elevated at any time, and smell apparently was not disturbed. Toward the last, in addition to the tendency to fall forward, and the pain in the neck, slight paresis of the left facial nerve had developed. Operation was not performed, chiefly on account of the objection of the relatives.

17. *Relapsing Bilateral Ptosis.*—Bychowsky reports the following interesting case. A girl of fifteen years gradually developed ptosis. In the morning she noticed that the eyelids felt heavy, and toward evening she was unable to lift them. The condition lasted three days when electricity was employed and she recovered. At the age of nineteen the ptosis again suddenly recurred. It was complete and was not accompanied by contractions, so that it was easily possible to raise the lids. The eye muscles appeared to be normal; the pupils reacted promptly; the patient's nutrition was good; there were no paralyses of the muscles of articulation, although the speech seemed to be slightly nasal. The patient was given galvanic current and felt better, although the ptosis persisted. Finally, however, during the night it suddenly disappeared. At the age of twenty-one a third attack occurred. On this occasion the patient had severe headache, but the

symptoms otherwise seemed to be the same as in the last attack. The electrical reactions of the muscles of the eye were normal. It was noted, however, that the muscles of the arms became very rapidly fatigued upon exertion, so that after lifting them five or six times they were practically paralyzed. During the application of the electricity to the muscles of the face the ptosis suddenly disappeared and there was not myasthenia of the muscles of the eye; it persisted, however, in the muscles of the arms. In the course of four days, however, this had also vanished. Bychowsky gives a careful discussion of the literature of myasthenia and recurrent paralysis, and reaches the conclusion that his case belongs to the group of myasthenic paralyses. It also bears a certain relation to the *Vertige paralyssant* of Gerlier, and the *kubisagari* of Miura, probably identical conditions. In a postscript he mentions the fact that at the age of twenty-two the patient had a fourth attack during which Bychowsky was able to determine the presence of the myasthenic reaction in the *ocularis* and *biceps*, and the extensors of the fingers on both sides. This reaction persisted for a time and then the patient improved.

18. *Cystic Meningitis*.—Rosenblath reports the following case. A man of forty-seven years was suddenly attacked by trembling of the limbs so that he was obliged to sit down. This continued for two weeks and rendered him incapable of even dressing himself. Later he began to have headache on the left side, was depressed and stopped work. A careful examination failed to show any changes in the motility, sensation, or the reflexes. Finally the patient complained of inward tremor; there was no obvious oscillations of the limbs. Later he developed extremely severe headache and pain and stiffness in the neck. Then there were attacks of vertigo causing him to fall down. It was then observed that he had choked disk. The forehead was tender to percussion, but motility, sensation and the reflexes were normal. There was neither ataxia nor tremor. The attacks of vertigo were sudden, causing him to fall heavily although he rarely lost consciousness. His intelligence gradually altered, his memory was impaired, and occasionally he seemed confused. Sometimes he had hallucinations of vision. His mind gradually failed; he developed *agraphia* and *alexia*, became more and more apathetic, and toward the end had vomiting. There were some anesthesia in the right half of the body; his temperature rose, he had a tendency to sleep, and finally died of dyspnea, and paralysis of the muscles of deglutition. At the autopsy some calcareous infiltrated cysts and some clear cysts were found in the neighborhood of the chiasm at the foot of the third frontal convolution on the right side; at the top of the temporal lobe and over the pons and medulla. A large cyst was found also in the posterior portion of the left lateral ventricle. A few cysts were also found in the upper portion of the cervical enlargement. Around these cysts there was occasionally cellular infiltration, particularly in the neighborhood of the blood vessels. A diagnosis of *cysticercus racemosus* of the brain was made. It is probable that the symptoms were produced rather by the involvement of the meninges and the blood vessels in the neighborhood of the cysts than by the cysts themselves. Rosenblath believes that *cysticercus racemosus* of the brain is not so very rare; death usually occurs slowly, the symptoms lasting one or two years, although they may last longer; and Zincker has believed that in two of his cases the symptoms persisted for seventeen or eighteen years. The earliest symptoms are those of *neurasthenia*; later there is weakness of the limbs, pain and stiffness of the neck, then attacks of weakness associated with tremor of the extremities; headache and vertigo are quite common. Cramps have been described in about half of the recorded cases; they may be slight twitchings of the arms

and legs, or they may resemble epilepsy. Not infrequently paresis may occur. The eyes may become prominent, there may be diplopia or ptosis. In many cases there is sudden transient blindness, and in a few hallucinations of vision. Aphasia is rare. Cerebral vomiting is very common. As a general rule patients become demented, but occasionally they are maniacal. It is possible that many of these symptoms are due to vital phenomena on the part of the cysticercus, although it seems to be certain that the majority are dependent upon changes in the blood vessels. It is most difficult to distinguish this condition from cerebral syphilis.

J. SAILER (Philadelphia)

JOURNAL DE NEUROLOGIE.

(1903, VIII, No. 1, January 5.)

1. Methods of Improving the Organization of the Belgian Asylums. J. CROCO.

1. A discussion of the method of caring for the insane in Belgium and in other countries, with suggestions as to their improvement, which is not suitable for review here.

(1903, VIII, No. 2, January 20.)

1. Facio-scapulo-humeral Hemiatrophy. A. DEBRAY.
2. Case of Hystero Syphilis. THOORIS.
3. Note on the Babinski Reflex. C. HELDENBERGH.

1. *Facio-scapulo-humeral Hemiatrophy*.—A report of a case of this character beginning subsequent to fracture of the wrist. The author thinks that the long continued sensory irritation due to the wrist injury may have sufficed to determine a functional disturbance in the trophic nerve cells situated in neighboring medullary segments, hence the atrophy. There was paradoxical electric reaction in the atrophied muscles, faradic irritability and, to a less extent, galvanic irritability being increased. There was some improvement under the continued use of faradism with rubbing and suitable diet.

2. *A Case of Hystero Syphilis*.—Report of a case of hemiplegia and hemianesthesia, in a man of twenty-seven years of age, formerly syphilitic, in which the combination of symptoms, and the lack of effect from syphilitic treatment, caused the author to conclude that the trouble was of hysterical, and not of organic origin.

3. *Note on the Babinski Reflex*.—A short discussion of the method of production of this reflex which the author thinks the type of abnormal cutaneous reflexes, an extrapyramidal, or "spino-mesencephalic" reflex, as he calls it.

(1903, VIII, No. 3, February 5.)

1. Multiple Sclerosis. BOUCHARD.
2. Bilateral Atrophic Paralysis of the Tongue. DECROLY

1. *Multiple Sclerosis, Movements During Repose*.—Description of a case observed during six years (from the age of sixteen to that of twenty-two years), which presented the following sequence of events. At first headache, vertigo, drooping of the upper lids and deviation of the right eye outwards, next loss of power, and exaggerated reflexes in the lower limbs, with continuous involuntary movements in them, these later being replaced by similar movements in the upper extremities. Speech then became difficult, of a scanning character, and finally unintelligible, while the legs grew so stiff as to make walking impossible. These symptoms, which took a year for their development, remained stationary for a long

time, and then began an improvement which proceeded to a remarkable degree, to "almost a cure." The patient's mental condition, however, remained defective. The author discussing the diagnosis considers especially Friedrich's disease, cerebellar heredo-ataxia, Parkinson's disease and multiple sclerosis. He concludes that while involuntary movements during repose are rare in the latter disease—a few cases of the sort have been reported—the clinical picture upon the whole corresponds best to that of multiple sclerosis.

2. *Suboccipital Vertebral Disease, with Bilateral Atrophic Paralysis of the Tongue.*—A description of a case of this character which began with pain in the head and neck, paralysis of the left arm and leg next appearing, and extending later to the right arm and leg. The man coming under treatment at this time extension, on the inclined plane, was made with so much improvement that he was able to get about and to work again. The bone disease progressing, however, the head became gradually displayed backward, and the tongue underwent complete atrophy, probably from pressure upon both hypoglossal nerves. Next, in spite of treatment, the paralysis of the limbs again increased, and the patient had become completely bedridden at the time of the report.

(1903, VIII, No. 4, February 20.)

1. Differential Diagnosis of Epilepsy. KOWALEVSKY.

2. Sarcolysis in Muscular Regression. DEBUCK and DEMOOR.

1. *Differential Diagnosis of Epilepsy.*—Will be abstracted when completed.

2. *Sarcolysis in Muscular Regression.*—Muscle is composed of two distinct morphological elements. (1) The sarcoplasm or non-differentiated protoplasm, with the nuclei, the nutritional element. (2) The myoplasm or differentiated protoplasm, the functional element.

The first is the one reacting to morbid agents. From the study of numerous preparations the authors are convinced that the muscle fiber plays an active rôle in its own regression, that it tends to return to an embryonic plasmodial, and cellular state, and that in this process the nuclei exercise an active biochemical or hystolitic influence, a phenomenon which they call "sarcolysis." The muscle cells play in this process a phagocytic rôle. These "sarcolytes" they think after repassing the embryonic stage can undergo a metaplasia, being transformed into connective tissue, and in this way a great part of the connective tissue, which replaces atrophied muscular fibers is produced, a "muscular autosclerosis."

ALLEN (Trenton).

THE
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Original Articles.

THE SIGN OF THE ORBICULARIS IN PERIPHERAL FACIAL
PARALYSIS.¹

BY DR. GEORGE W. JACOBY.

The divergence of views as to the course of the central neurones of the facial apparatus has always been very great.

All of our knowledge had been derived from pathologico-anatomical, and experimento-physiological observations; clinical study failed to cast any material light upon the subject, because all deductions from clinical observations were dependent upon the assumption that in facial paralysis due to central affections, the temporo-facial branch of the facial nerve was not involved, and that in ordinary hemiplegia the upper part of the face entirely escapes being paralyzed.

In order to explain this non-involvement of the upper facial, certain older authors questioned whether the upper facial had any cortical origin whatsoever, and believed that its only central source was from the nucleus of the fourth ventricle. Others (Huguenin, Chvostek and Hallopeau), while admitting a cortical origin, assume that the upper facial reaches the facial nucleus from the cortex by means of a course, separate from that of the lower facial; while still other and later observers have assumed that separate centers exist in the cortex for the upper and lower facial, that for the lower facial lying in the lower extremities of the frontal convolutions, while that for the upper

¹Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

facial was variously assumed to lie in the second frontal convolution at its junction with the precentral (Ferrier, Horsley, Hitzig and Duret), or in the inferior parietal lobule (Mendel, Exner and Paneth, and Obersteiner).

More recent clinical observations have, however, shown that the assumption of non-involvement of the upper facial in hemiplegia is a mistake. The recognition of this long dominant error in clinical knowledge is due in great part to the sign of the orbicularis (*Signe de l'orbiculaire de la paupière*). This name was first suggested by Révilliod to describe a symptom which was already known but which he above all others has most carefully studied and utilized for scientific deductions.

This symptom consists in the inability of a hemiplegic, voluntarily to close the eye upon the paralyzed side, except in conjunction with the other eye.

Thus in cases of hemiplegia, the temporo-facial branch of the facial nerve is often so slightly affected as to lead to doubt whether the upper facial territory is involved at all.

If in such a case the hemiplegic be asked to close both eyes together, he is able to do so without difficulty; he is also able to close the eye upon the sound side while keeping the eye upon the affected side open, but he is unable to reverse this procedure and close the eye upon the paralyzed side while keeping that upon the unaffected side open.

This sign of the orbicularis was found by Révilliod as well as by subsequent observers, Boiadjiew, and Bard, to be present in almost all hemiplegics, and this not only for a short time after the apoplectic attack, but frequently persisting for a long time after all other signs of paralysis have passed away.

Every subsequent investigator has been able to corroborate these earlier observations, so that as a result of this, and other clinical signs which do not concern us here, it is today universally admitted that the upper facial is involved in all central affections of the facial nerve, although to a less extent than is the lower facial.

This being acknowledged, it is also clear that the various assumptions regarding the dissociate cortical origin of the upper facial are superfluous and the most modern authors agree with Mills who sums up the central course of the facial as follows:

"The central neurones of the facial apparatus arise from the motor cells of the lower extremities of the central convolutions. Together they form a tract which passes by the corona radiata and the internal capsule about its knee to the tegmentum, decussating in the latter to enter the facial nucleus of the other side."

If, however, the implication of the upper facial in the central paralysis is a generally recognized fact, it must be admitted on the other hand also that the upper facial in such paralysis is always relatively free, that it is rarely as much involved as the middle and lower facial or as much as the upper facial is involved in peripheral paralysis.

This fact requires explanation!

Why is the upper facial relatively free in central paralysis?

A priori, it may be reasoned, the functions of the upper and lower facial territories differ materially, the upper being utilized more for emotional movements, while the middle and lower serve more for voluntary ones; that emotional movements are as a rule bilateral, while voluntary ones may be either unilateral or bilateral; that, furthermore, inasmuch as the voluntary movements in cerebral hemiplegia due to lesion of the pyramidal tracts, are the ones most affected, it will be comprehensible that the middle and lower facial muscles will be more markedly involved than those supplied by the upper branch of the facial nerve.

This reasoning presupposes a special path, not through the internal capsule, for emotional movements, or it warrants the assumption that each upper facial is innervated from both hemispheres. While the first assumption is by many considered correct, it would, however, in the case of cortical or subcortical lesions (that is, involvement of the purely voluntary motor paths), necessitate equable paralysis of all parts of the facial, which is contrary to all clinical experience.

The other assumption was first advanced in a general way by Broadbent, and is almost universally accepted as a fact.

Broadbent's hypothesis in the main represents the following ideas: Many muscles of the body are used only in conjunction with their fellows of the opposite side; others while usually used alone may be and frequently are used conjointly with their fellows. Those muscles which are always used alone are represented only unilaterally in the cortex; those that are used conjointly are bilat-

erally represented, so that unilateral movements can be excited only from one, the opposite hemisphere, while bilateral movements can be excited from either hemisphere, and finally those muscles which are sometimes used alone and at other times conjointly are connected with both hemispheres, but are generally excited from one, the opposite hemisphere, only.

The synergic action of the orbiculares palpebrarum, during repose or during a state of unconsciousness, shows that they must have a bilateral innervation and that they must, therefore, be excitable together from either hemisphere. As a result of education and under influence of the will each orbicularis may in its action become independent of the other, and must then be excitable individually from the contralateral hemisphere. Such conjoint and independent actions can be explained only by the assumption of a chiasm formation, an interlacement composed of two crossed and two direct tracts. Therefore Révilliod, starting from the hitherto accepted premise that the sign of the orbicularis is never present in peripheral paralysis, but is found only in central affection, seeks this chiasm formation at a point on the motor path between the cortex and the nucleus. Révilliod says: "If indeed, as the sign of the orbicularis has demonstrated, the superior facial is really implicated in ordinary hemiplegia together with and by means of the same focus as the lower facial and the extremities, then it must form part of the same tract, called pyramidal, and course with it from the cortex to the oblongata, passing by way of the internal capsule. But in order to assure the synergy of the two sides of the upper face, this nerve must divide before reaching its nucleus, into two branches, the one crossed, the other direct, the latter returning to the face upon the same side as the hemisphere from which it emanates," and "the crossed branch would consequently always be accompanied and assisted into its end ramifications, by the direct branch which comes from the hemisphere of the opposite side." This explanation Révilliod illustrates by a diagram of the motor tract in which the decussation of nerve fibers is shown to be complete for the lower facial (and the extremities) but is assumed to be incomplete for the synergic muscular groups of which the orbiculares form a morphological part.

Révilliod's assumption may be endorsed but at the same time may be considered insufficient.

If we recall that the upper facial also in other pathological conditions than that of central hemiplegia may show a comportment different from that of the middle and lower distributions; that in bulbar paralysis it is almost the rule to find the upper facial relatively less implicated than the lower facial, and that even in purely peripheral facial paralysis the temporo-facial branch is not infrequently much less affected than is the lower facial, then we are entitled to assume, not only as has been done by others, that the upper facial is especially resistant to pathological influences, but also that a decussation of fibers must take place at the facial nucleus itself and that this decussation is mainly made up of those fibers which act synergically.

This question of decussation of facial fibers is one which anatomical and physiological investigations, as well as pathological studies, have been unable to answer definitely.

A priori, and reasoning from analogy it would seem that such decussation must exist. Clinically we know that in complete hemiplegia, not only is, as has already been emphasized, the orbicularis palpebrarum relatively free, but also that the eye muscles, the muscles of the trunk and of the larynx, in short all those muscles which usually functionate in pairs and give rise to bilateral associated movements are comparatively unaffected.

This can be explained only if we admit the existence of commissures joining the nuclei of origin of the motor nerves of the two sides, such commissure then assuring functional physiological synergy, and reciprocal subserviency in pathological implication of the nucleus upon one side.

As a general principle the existence of such decussation between the homologous nuclei of all motor cranial nerves is admitted (see Edinger), but it is not admitted as a fact by all observers for all cranial nerves.

Thus as regards the facial, we find that Adolf Meyer, C. Mayer, Van Gehuchten and Bischoff have furnished proof in opposition to the doctrine of decussation between its roots.

On the other hand, Obersteiner, von Bechterew, Flatau, Marinesco, Bruce, Kotelewsky, Mills, Pardo and Bary are sup-

porters and have furnished anatomical proof of the existence of such decussation.

Recent observations of Ramon y Cajal are also in accord herewith and clearly show that fibers belonging to the seventh nerve decussate in the raphè.

Of special interest and value in this connection, are the experiments of J. and N. Aspisow. These observers after having, by means of electric excitation, located the cortical center for the upper facial in dogs, excised this center and after proper lapse of time examined the brain.

In addition to secondary degeneration through the corona radiata, internal capsule, crus and pons, the degenerated fibers could be plainly traced from the homolateral, across the raphè, to the contralateral facial nucleus.

Against all this anatomical proof, however, stood the clinically accepted fact that the sign of the orbicularis was absent in peripheral paralysis. As a result of my examination of all the cases of peripheral facial paralysis which I have seen in hospital and private practice, since the last four years, I am able now to say that this so-called fact is an error, and that the sign of the orbicularis can be found in many peripheral cases if sought for at the proper time.

Already in 1894 Mann, in calling attention to the fact, which had also been recorded by a number of others, that the orbicularis oculi in some cases of peripheral paralysis is not nearly so much involved as the other facial muscles, and that this same muscle in cases which are improving shows an early regeneration, also states that in these same cases the patients were able to close the eyes at a time when the attempt to corrugate the forehead failed; the forehead upon the paralyzed side remaining smooth but the eye closing.

If this investigation be probed further, it will be found that while many such patients can at this time close both eyes together, the independent closure of each eye is possible only for the eye upon the unaffected side.

Before this test can be utilized for deductions or conclusions of any kind, it must, however, be first shown that the independent closure of each eye exists physiologically in the majority of individuals. This has been demonstrated by Boiadjew, who tested

750 healthy persons, and found that 484 or 64.54 per cent could close each eye independently; 126, or 16.80 per cent, could close only the left eye without at the same time closing the right; 64, or 8.53 per cent, could close only the right eye without at the same time closing the left; 76, or 10.13 per cent, could close neither the left nor the right eye independently of the other.

This investigation, therefore, shows that two-thirds could close either eye independently and that, therefore, this capability is sufficiently frequent to allow its absence to be utilized as a clinical symptom, provided the patient can give a history of previous independent control.

There is no necessity for giving a large number of cases in order to demonstrate the frequent occurrences of a symptom which any observer may easily corroborate. Let us take a few typical cases which present points of special interest.

Thus, W. W. O., male, aged thirty-seven, was seen on October 16, 1899; he had then been afflicted with a peripheral paralysis of the right facial nerve since a few days. There was marked lagophthalmus, with inability to corrugate the forehead, complete R. D. in all the muscles supplied by the facial nerve, including the orbicularis oculi; no disorder of taste nor of hearing. Ten days after onset of the paralysis, with R. D. still existing in all the facial muscles, including the orbicularis, and while still unable to corrugate his forehead, to whistle or to blow out a light, he could close both eyes together, but could not close the right eye independently of the left. He could close the eye upon the sound side, alone; he could close the affected eye when the other eye was closed, but he could not close the affected eye alone when the unaffected eye was kept open.

Two weeks later he was able to close each eye freely and independently of the other, as he had always been able to do prior to the onset of the paralysis.

The following case shows the long persistence of an untreated facial paralysis, the improvement comparatively soon after treatment was instituted and the supervention of the sign of the orbicularis contemporaneously with the onset of improvement.

A. L., a young boy, was operated for mastoiditis; after coming out of the narcosis it was noted that he had a complete left-sided facial paralysis. For one and one-half years he was subjected to

much treatment on account of his ear trouble, but no attention was given to the facial paralysis. At the time when I first saw him he still showed complete paralysis of the entire facial with marked R. D. in all the muscles supplied by this nerve, no faradic response being obtainable in a single muscle. After five months' electrical treatment, faradic reactions set in in the temporo-facial as well as in the lower facial, but not so marked here as there; at the same time functional improvement took place in the upper facial alone and he was soon able to close the eye completely. He could, however, not close the affected eye without also closing the sound one.

Four weeks later, while still showing R. D. in the entire upper and lower facial territory, he was able freely to close each eye separately.

The mother states that prior to the facial involvement he was able to wink each eye alone.

The last case which I will briefly relate is of interest because it was a case of peripheral facial paralysis with apparent freedom of the orbicularis, which, however, through the presence of the sign, was shown to be an ordinary case with but slight involvement of the upper branch.

The patient, a middle-aged woman, was seen in March, 1900, three days after the acute onset of a right-sided facial paralysis. Examination showed marked involvement of the lower facial, as shown by the interference with mouth movements (whistling, blowing, grinning, etc.), with apparent freedom of the upper facial. She was able to close both eyes completely and easily. When each eye was tested separately it was, however, found that she could close the left eye alone with ease, but that she could not close the right at all except in conjunction with the other eye.

She made a rapid and complete recovery and was then able to close each eye singly as she had been able to do prior to the onset of the paralysis.

Without citing any more cases I desire to call attention to a result of my investigation which seems to me to bear some prognostic significance. As well as it has been recognized that in peripheral paralysis of the facial nerve, the orbiculo-frontal branches are frequently less implicated or recuperate earlier than the other branches, so also it is well known that marked spasm and

twitchings may occur in the orbiculo-frontal territory, at a time when the voluntary path is being reëstablished, and at a time in which reduced electrical excitation still exists. In what percentage of cases such spasms occur, I do not know, but the number is probably not small; I am, however, able to state with certainty that in no case which I have followed during the last four years has such spasm occurred after the sign of the orbicularis, which had been present, had passed away; or I may say in all cases in which the spasm has occurred the patient was not able to close each eye independently at the time of the onset of the spasm.

In conclusion I would summarize the result of my studies as follows:

1. The statement of Bard, that the sign is invariably lacking in peripheral facial paralysis and is, therefore, a distinguishing mark of central affection, is incorrect.
2. The presence of the sign in peripheral paralysis is further proof of the existence of commissural fibers between the nuclei of the 7th nerves.
3. The sign is of clinical value in so far as its presence in peripheral paralysis shows that complete recovery has not yet occurred.
4. The secondary over-action in the orbicularis palpebrarum, which is late in appearance and always coincides with some recovery in power, does not occur in those cases in which the sign having been present has passed away.

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GIGANTISM AND LEONTIASIS OSSEA, WITH REPORT OF THE CASE OF THE GIANT WILKINS.

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(Continued from page 532.)

In view of the findings in this case one must admire the keenness of Sternberg's judgment in excluding acromegaly and pointing out the analogy with Buhl's case (to be related later). The relation of acromegaly to gigantism, and of both to disease of the hypophysis, has been extensively discussed of late. Thanks to this fact a number of exhaustive reviews of the cases of all more or less scientifically studied giants exists. The most thorough ones are those by Sternberg (43), Meige (31), and Woods Hutchinson (27). All authors agree that giants frequently present acromegalic symptoms, and that hypophysis disease is the rule in acromegaly. A most daring generalization is that of Massolongo (30), who says: "Acromegaly is nothing but delayed, abnormal gigantism." Meige quotes and indorses this statement and gives the easy explanation that, when the disease commences in youth we get a case of gigantism; when in adult life, acromegaly; if commencing in youth and continuing into adult life we get a combination of the two. After an extensive review of cases* he formulates the following statements (*loc. cit.* p. 458): "1. Acromegaly never precedes gigantism. 2. Acromegaly sets in during the course of about one-half of the cases of gigantism. 3. When acromegaly is associated with gigantism, the latter always appears first." Woods Hutchinson sums up the result of his studies of gigantism as follows: "1. The greater part of the overgrowth is found at or near the tips of the segment-crescents, as in acromegaly, differing from the latter mainly in that it is not exclusively confined to the tip of the segment or last division of the limb.

*Among these our Wilkins appears as two, the author having failed to observe that Dana and Lamberg described the same man.

2. The facial part of the skull is enlarged out of all proportion to the cranial, particularly in the region of the lower jaw. 3. The condition, whether it be regarded as normal or morbid, is one that distinctly tends to shortness of life, and would appear to have an average duration of scarcely more than twenty years. 4. The mental and physical vigor of the giant is distinctly below par, and his death usually comes either from a steady progressive increase of this weakness, or from some trifling accident, or usually mild, intercurrent disease. 5. Sexual powers appear in the great majority of cases to be far below normal. 6. There is a decided preponderance of males among the victims of this condition; in all of which statements there is a decided parallelism with acromegaly. Last of all, and from the point of view of this essay, of greatest interest, is the fact that the one morbid condition which is peculiar to both of these disturbances of nutrition, the enlargement of the pituitary body, is found to be present in a large majority of cases of both." After stating that the material as yet is meager, he says that we are "justified at least in the tentative conclusion, until some evidence to the contrary can be adduced, that acromegaly and gigantism are simply different expressions of one and the same morbid condition; in other words, that acromegaly is a general overgrowth tendency which does not, for some reason, begin to express itself until after adult stature has been reached, and which consequently expends itself upon those points in the body at which growth last ceased—the extremities of the segment-crescents and the distal extremities of the appendages. Second, that gigantism in a large majority of cases is this same condition manifesting itself in childhood or before complete stature has been reached, and the growth in consequence is more symmetrical and less strictly confined to the last segment of the arches and appendages. In most cases, however, the tendency appears to be for these last segments to grow in an unsymmetrical and excessive manner." After a discussion of the conditions found in the hypophyses of acromegalics, giants and dwarfs, he further concludes: "1. That the pituitary body is still functional. 2. That disturbances of its metabolism are the principal factors in both acromegaly and gigantism, the difference between the results being simply due to the stage of individual development at which the disturbance of the function begins. 3. That the nature of the

overgrowth in both these diseases is primarily on the order of a pure functional hypertrophy; later, however, losing some of the definiteness of its impulse, and either producing immature tissue of a mixed type or resulting in simple hemorrhagic exudation, with either cyst formation or complete breaking down of the tissue mass. 4. That it seems probable, although upon this head the evidence is still uncertain, that some part is played by this body in 'dwarfism,' rickets, and the dwarf forms of cretinism. 5. That a reflex disturbance of its function may possibly underlie the dystrophy accompanying pharyngeal adenoids. 6. That it would appear to be a sort of 'growth center' or proportion-regulator of the entire appendicular skeleton." The author finally hints that chorea may be due to disturbance of the hypophysis, and promises a further report on the subject.

Sternberg (43) states that about 20 per cent of all acromegals are over 177 cm. (5 ft. 10 in.) tall, and that about 40 per cent of all giants have acromegaly, but denies the identity of the two processes which up to that time had been advocated by Klebs, Massolongo, Brissaud and Meige. He believes there are "normal" giants, i.e., well-proportioned over-grown people. As such he regards Virchow's case, Winkelmeyer (48), whose height is given as 227.8 cm. Hutchinson does not consider him "normal," as his height at the hip line, as determined by the position of his hand in the picture, is 64 per cent of the total height, instead of the normal 55 per cent. Sternberg classifies the "pathological" giants as follows: "1. Acromegals—about 40 per cent of all giants. 2. Cases with multiple tumor-like exostoses (leontiasis ossea and hyperostoses). Often the signs of compression of the brain and cranial nerves. Here he groups Buhl's case, Hasler (10), 227 cm.; Sirena's (42) first case, 240 cm., and our case, Wilkins, 245 cm. (Reference given to Lambert's report). 3. Cases with facial hemihypertrophy. Dana's case.* 4. Cases with multiple curvatures of the bones, as scoliosis, curved humerus, genu valgum, of similar appearances as in rickets; for instance, Bollinger's giantess (6) and Zitterland's case, Berlin skeleton 3040 (51). 5. Gigantism in hereditary syphilis, case of Fuchs (18). 6. Precocious development and rapid growth in cases of testicular tumors in children. Sacchi's case (38)."

*I suspect Wilkins is the case referred to. Reference is given to the article in which his case is reported.

Meige, who quotes this classification, finds it a little artificial. This may be true, but it probably goes into generalizations as far as the facts warrant at present.

Sternberg sees two possibilities for an etiologic relationship between gigantism and acromegaly. First, the latter may induce a cartilaginous proliferation in a still ununited epiphysis; or, secondly, in gigantism a disposition to general dystrophies, and particularly to acromegaly, may exist.

Schmidt (41) inclines to the latter view, and considers the acromegalic features as something complicating the gigantism, the same being true of the frequent scolioses, genua valga, etc., and the rarer hyperostoses of the bones of the head, as seen in our case, which is, together with that of Buhl, quoted as one in point (from Lamberg's report).

As Buhl's case (10) is in many respects similar to our own, it deserves special mention. It was that of a man, Thomas Hasler, who was normal up to the age of nine years, when a rapid growth commenced. This is said to have followed a kick on the cheek by a horse. Some years later huge hyperostoses of the facial and cranial bones appeared and were larger on the injured side. At twelve he measured six feet. He died at twenty-five. His height then was 227 cm., or 235 cm. (7 ft. 8½ in.) with the back stretched; the weight, 155 kg. There were no lesions of the viscera. All epiphyses, including those of the trochanters, were sharply outlined. The skull showed enormous exostoses. The lower jaw was enlarged in all directions, very irregular, its height at the middle of the chin 12 cm.; thickness, 9 cm.; the left side was the thicker. The upper maxillæ were also thick. On the left side the processus frontalis, the nasal, malar, ethmoid and lachrymal bones were greatly thickened, the floor of the orbit raised, the left wall of the nose displaced to the right. The left frontal bone measured 6 cm. (2¾ inches) in thickness, the left temporal, 1.5 cm.; the right, 1 cm. The left side of the occipital and parietal bones also thick. Both in the frontal and parietal bones superficial porous islands were found. The interior of the skull was diminished on the left side, the sella turcica displaced to the right. The brain weighed 1,465 grams, and was much decomposed at the time of the autopsy. The hypophysis

is not mentioned. Buhl attributed the patient's death to compression of the brain.*

Hutchinson's hypothesis that the hypophysis is a "growth center or proportion-regulator of the entire appendicular skeleton" is a bold one, considering how few actual facts we possess concerning the functions of that organ. Benda in quoting the above statement (4) says that Hutchinson is bolder than himself. The physiologists seem unable to agree about a single function of the hypophysis, and some deny its having any function at all. The studies of Andriezen (2) on the larval amphioxus, ammatetes, balanoglossus and other lower animals are very fascinating.



Fig. 10. Base of skull in Buhl's case.

He observed carmine solutions passing from the buccal to the neural cavity through the hypophysis and infundibulum. He says: "A water-vascular system permeating the body-tissues, and serving for the in-bringing of oxygen and the out-taking of waste products, and having inlets and outlets communicating with the aqueous medium in which the animal lives, preceded, in point of time, the development and elaboration of a true blood-vascular system." He believes that the anterior or glandular portion of the hypophysis in these embryos secretes a substance which modifies the fluid passing in from the buccal cavity, while the

*A glance at the picture of the skull will show the close resemblance to our case. As the original communication is difficult of access this drawing is reproduced here.

posterior or nervous portion controls that secretion. His predictions of the symptoms following extirpation are also interesting, as they were confirmed by the experiments of Vasalle and Sacchi (47).

Cyon has performed many experiments and written a number of articles on the physiology of the hypophysis. In the most recent one (12) he relates how he irritated the organ in various ways through an opening made in the pharynx. He claims the following functions for it: 1. (a) It serves to regulate intracranial blood pressure, (b) regulates metabolism. 2. It regulates cerebral pressure in two ways, (a) mechanically, each increase in pressure irritating it, causing slower and stronger heart beats and increased rapidity of venous circulation, particularly of the thyroid, thus relieving the brain; (b) chemically, as it produces substances, probably two in number, one of which irritates the vagus, the other the accelerators. 3. Effect on metabolism takes place through similar action of its products on the vagus and the sympathetic, seen in increased oxidation and decreased weight. 4. Tonic excitation of the vagus depends on excitation of the hypophysis due to pressure exerted on it. 5. Continued excitation, especially electric, of the hypophysis, causes as after-effects severe epileptiform spasms, most easily explained by circulatory disturbances in certain parts of the brain. In rabbits electric irritation caused persistent erections and increased diuresis; the latter also followed the injection of extract of the hypophysis. Cyon believes that the effect upon the circulation is due to a stimulation of thyroid activity, regulating the circulation through the carotids. Hypophysin, the most important substance obtained from the hypophysis, has an action similar to, but more intense than that of iodothylin. In thyroid disease the hypophysis may for some time take up the function of the thyroid. Thus, enlarged hypophyses were found in thyroidectomized rabbits, and the roof of the hypophysis cavity often ossified, which, according to Cyon's theory, ought to facilitate the function of the gland. But in spite of his conviction of the correctness of his views, Cyon is unable to explain acromegaly either by hyper—or hypo—function. Certain symptoms, such as impotence in men and amenorrhea in women, he interprets as due to a loss of function.

Enlargement of the hypophysis has been described as following thyroidectomy by Hofmeister (26), Stieda (45), Gley (19), Goldberg (20), and others; in cretinism by Bourneville and Bricon (7), Dolega (14), Osler (36), Grön (21), and others. Le Count (32) states that in acromegaly the thyroid has been recorded as reduced in size four times, as cystic or the seat of colloid degeneration, twice, and as normal in appearance five times. Ponfick (37) reports two cases of myxedema in which the glandular atrophy and increase in fibrous tissue were so much more marked in the hypophysis than in the thyroid that it seemed probable the change had begun in the former organ, which Ponfick suspects may be closely related to the production of myxedema.

Friedman and Maas (17) disagree with Cyon's views, and from a number of extirpations of the hypophysis conclude that it is not essential to life, and state that, in their experiments, its absence led to no change in any organ, particularly not in the thyroid.

Castelli (11) gives as the effects of extirpation: More rapid heart action, slower respirations, psychic depression, tetanic-clonic cramps, and cachexia, ending in coma and death. He believes it is concerned in the rendering harmless of toxines arising in the organism—which is, in turn, denied by Cyon.

Biedl and Reiner (5) also attacked Cyon's views, and claim that electric irritation of most places of the brain has the same effect as when applied to the hypophysis.

With such lack of definite knowledge as to its functions, it is no wonder that the rôle played by the hypophysis in disease can only be guessed at for the present. No one can deny the fact that it is diseased in most cases of acromegaly and in many giants, but whether this is the cause or merely itself a phenomenon of the disease cannot be stated. It is also probably true that the majority of the hypophyses tumors observed in acromegaly are to be considered as hyperplasia of the glandular portion. They have been given a great variety of names, but as Benda (4) well puts it, the names given differ more than the descriptions, and the histological picture in a given case may vary so much that one may find microscopic fields to fit almost any tumor name. In four such cases Benda succeeded in demonstrating glandular

structures and transitional forms between these and the irregular sarcoma-like portions found. In the present case it was confidently expected to find such transitional forms, and not until the serial sections through the hypophysis had demonstrated the existence of a sharp border at every point, was the glandular origin of the tumor definitely abandoned.

Many cases of hypophysis disease without acromegaly or other disturbance of growth are on record, as Handford's case (22), Woolcombe's case of psammoma (49), Beadle's two cases of gumma and tubercle respectively (3), Breitner's of adenoma (8). In this country Hektoen (23) has reported a case of gumma associated with a thick skull, Stewart (44) and Hinsdale (25) of sarcoma with no acromegalic symptoms. Recently Taylor and Waterman (46) reported a case of a sarcoma involving the optic commissure, but its connection with the hypophysis was not proven, and there is no mention of an examination of the sella turcica after the removal of the brain. In an autopsy on a demented female epileptic, aged 38 years, at the Iowa State Hospital, Mount Pleasant, the writer found an hypophysis that weighed 59 grams. The skull, particularly the frontal bone, was thick. The brain weighed 1,040 grams. The woman was very small and had no acromegalic symptoms. In an insane, tuberculous subject, likewise with no acromegalic features, a small calcareous nodule was found in an otherwise normal hypophysis.

The following instance of gigantism, associated with a tumor of the pineal body, while the hypophysis was normal, is of considerable interest. It is reported by Oestreich and Slawyk (35). A male child had been delivered by forceps and been long asphyxiated. There were spasms of the glottis at the age of three months. The child developed normally the first three years, then it became morose and the body began to grow rapidly, particularly the penis. Walking became difficult. July 4, 1898, four years old, it was admitted to the Berlin Charité. Height 108 cm., weight 20 kg., muscles and skeleton powerful. Its size was that of a normal child of seven or eight years. Skin normal. Head symmetrical, circumference 53 cm. (4 cm. above normal, according to the authors). There were sluggish reaction of the pupils to light, slight convergent strabismus, and a bilateral choked disc.

Sight appeared impaired. Mammæ, hypertrophic; penis, 9 cm. long; pubic hair, 1 cm long. Extremities of natural length. Sensations normal. After three weeks of clonic convulsions the child died Aug. 12, 1899. An autopsy was held. Anatomic diagnosis: 1. Cystic psammo-sarcoma of the pineal gland. 2. Internal hydrocephalus. 3. Gigantism. The skull was irregularly thinned. The posterior part of the third ventricle was occupied by an irregular, partly cystic tumor, the size of a small apple, in the position of the pineal body. The tumor also shows firmer parts and sand-like areas. The thyroid and hypophysis were normal, the thymus of proportionate size and normal histologically. The tumor was found to be a spindle-celled sarcoma, and the authors consider it the cause of the giant growth.

In spite of the title of the article, "Gigantism and Tumor of the Pineal Gland," and the explicit statement that the hypophysis was normal, Meige, in his previously quoted article (p. 472), in referring to this case, says that Oestreich and Slawyk described an acromegalic giant in whose *hypophysis* a tumor was found!

The presence of calcified patches in the spinal meninges of our case is of some interest. The deposit of externally generally smooth, internally rough flattened plates of bone on the inner surface of the dura has been designated "arachnitis ossificans" (40). According to Zanda (50) the bone formation takes place from the dura after adhesions with the arachnoid have been formed. They are most frequent in connection with regressive changes, such as those of old age, chronic insanities, and diseases of the spinal cord. Sainton and State (39) describe a case of acromegaly in a man 54 years old, with numerous such sand-paper-like patches enveloping the cord in the thoracic and lumbar regions, and considered them the cause of the cramps and pains in the extremities present. Similarly located bony or bone-like patches in acromegalics have also been described by Duchesneau (15), Henriot (24), and Finzi (16). In our case, it will be remembered, calcareous nodules were also present in the pleura and peribronchial lymph glands. Von Moraczewski (33) has shown a tendency to retention of lime and phosphorus in acromegalics, and Meige sees here a chance for a new proof of the identity of acromegaly and gigantism, if such a condition is found in the latter, as we might then presume that in the growing giant the osteogenetic function

is exercised only at the epiphyseal junctions, while, after the cessation of growth, the osseous hyper-function leads to the formation of aberrant calcareous deposits.

Heredity does not seem to play a significant part in the etiology of gigantism. In most cases the other members of the family are reported of ordinary stature and in good health. Thus "Lady Aama," the French giantess reported by Hutchinson (28), was the youngest of fifteen children, all the others being normal. If the sources of information of De Neuville (34) were even approximately reliable, a tendency to giant growth has existed in certain families. According to him, Robert Hales, an Englishman born in 1820, measured 7 ft. 6 in., his father 6 ft. 6 in., and his mother 6 ft. An ancestor who lived at the time of Henry VIII measured 8 ft. 8 in. Five sisters averaged 6 ft. 3 in.; the tallest, 7 ft. 2 in., died at 20 years of age. Four brothers averaged 6 ft. 6 in. Hales himself measured 62 in. around the chest, 64 in. around the waist. Louis Frenz, born 1800, measured 7 ft. 6 in. He said one sister was "nearly as tall," and one brother "still taller." Achard and Loeper (1) report the case of a male acromegalic giant, height 212 cm. (6 ft. 11 in.) whose father measured 195 cm., paternal uncle 210 cm., and a sister 180 cm. According to Meige, the Chinese giant Chang, himself 8 ft. tall, said he had a sister 8 ft. 4 in.

Nearly all giants have been poorly developed sexually. With the possible exception of Winkelmeyer (Virchow's "normal" giant) none have left children, and all of them have been short-lived. Hutchinson gives the average age at death in eight giants as 21.3 years, the extremes being "in childhood" (the tall girl of Basle) and 25 years (Buhl's case, Hasler). According to Meige, the Chinese giant, Chang, must have reached 51 years.

The changes in the skull in our case are difficult to understand, partly on account of the complicated condition, partly because of the small number of analogous cases and the incomplete descriptions to be found of them. While there are many good gross descriptions and accurate measurements, histologic examinations have rarely been made. An attempt will be made here to review briefly the more important cases of cranial hyperostosis which are likely, at least in part, to be of a similar nature.

In 1697 Malpighi (69) described a skull weighing 3,618

grams, with effaced sutures, a tumor of cherry size near the sagittal suture, and here and there small marble-like eminences. The temporal bones were particularly large, the base of the skull thick, the orbits small.

Tarin (89) in 1753 described a thickened frontal bone with prominent angular processes.

Jourdain (67) and later Ribel (77) described the case of Forcade, the son of a French surgeon. He was well up to the age of 12 years when an abscess at the inner canthus of the right eye was opened and discharged for a long time. During this time an almond-sized prominence appeared on the nasal process of the right superior maxilla, and increased in size so that at the age of 15 years the boy was unable to breathe through the nose on account of it. Then, the lower jaw, nasal and malar bones, and floor and sides of the orbit became involved. Later, exophthalmos, blindness, and speech troubles appeared. The patient, who was below middle stature, died at the age of 45 years. The dried skull weighed 8 lbs. 3 oz., the lower jaw alone $3\frac{1}{2}$ lbs. There were huge exostoses of ivory hardness on the lower jaw and on the inferior margin of the orbits. The bones of the cranial vault were thick, with flat exostoses and very hard, the frontal and maxillary sinuses obliterated; cranial cavity not diminished in size. The facial muscles were fibrous in appearance and adherent to the periosteum. The bones outside the skull were unusually delicate.

Sandifort (78) described a skull thickened by about one inch with abundant spongy substance between the two thickened tables. On the interior, especially of the frontal bone, were numerous rough projections.

Jadelot (66) described and recognized as pathologic a skull which a previous writer (Wendelstedt) had contended must have belonged to a preadamite giant, at least 13 ft. tall, and which Cuvier, from the condition of the teeth and sockets, in turn had shown to have been that of a child of six or seven years. It weighed 3,913 grams, measured 236.9 mm. in length, 151.1 mm. in height, and 203 mm. in biparietal diameter. The thickness varied from 19.8 to 40.2 mm.; the diploë was more compact than the outer and inner plates.

Saucerrotte (79) describes a man, 39 years old, the circumference of whose head was 797 mm.; the lower jaw, the hands, feet,

spine, ribs, sternum and clavicles were large, with hyperostoses. The lips were thick. The position of this case is somewhat uncertain. Virchow, Paget, and Marie each claimed it for the diseases named by them, leontiasis ossea, ostitis deformans, and acromegaly respectively. Ilg (65) and later Gruber (61) report a case whose skull is the now famous one of the Prague Anatomic Museum. A girl of ten years, previously healthy, was taken with amaurosis, followed by severe headaches, delirium and epileptiform attacks. At sixteen she became deaf, at seventeen unable to swallow solid food, lost the sense of smell, and became bed-ridden. During this time she had several attacks of erysipelas. She died in 1804, 27 years old. The dry skull without the lower jaw, teeth and nasal bones weighed 4,200 grams. The fronto-occipital diameter was 197.6 mm., the biparietal 171.2 mm., the thickness varied from 19.8 to 52.6 mm., the occipital bone being the thickest. The external surface was uneven, the sutures had mostly disappeared, the prominences, particularly the external occipital protuberance, were enlarged. There were compact exostoses on the inner side of the frontal bone and greater wing of the sphenoid. The sella turcica was unusually shallow and narrow, and all foramina diminished in size. Diploë was only present in the frontal and parietal bones, in other places replaced by hard bone. The capacity of the cranial cavity was 1080 c.c. The specific gravity of the bone was 1.94. Microscopic examination: "The medullary canals narrow, surrounded by concentric bony lamella, mostly without sharp borders, the peripheral lamella darker than normal. Ground substance fibrillar, darker, with numerous powder-like dark granules. Bone corpuscles not clearly outlined, globular, or at least more globular than spindle-shaped; there are also some abnormally large spindle-shaped corpuscles. The ductuli chalicophori increased in size and number, with fewer anastomoses than normally." This is probably the first histologic examination ever made of a hyperostotic skull.

A very thick skull in Spurzheim's collection was described by Kilian (68). Its thickness was from 27 to 54.1 mm., latter in parietal region.

A still thicker one was found near Münster and recorded by Bojanus (54). Its weight without the lower jaw was 4,000

grams, its thickness 27-67 mm. Most sutures were obliterated, no diploë present. The orbits narrow, their walls thickened.

Breschet (55) reports a congenital case in a child who died at the age of 18 months in a tetanic spasm after having suffered with convulsions from birth, with only brief intermissions. All the cranial bones, except at the base, were thickened, in places measuring one inch. The anterior fontanelle had not disappeared. Bones of the face not involved.

The skull in Vrolick's (91) case weighed 1,305 grams, measured 199 mm. in length, 150 mm. in width, rough externally. The sutures, with the exception of the squamous, were obliterated. The frontal bone was 15-20 mm. thick, occipital 20 mm., right parietal 7 mm., left 15 mm., sella turcica narrow and deep.

In 1851 Albers appeared with a monograph on the subject (52), in which he denied the inflammatory nature of cranial hyperostosis and any connection with rickets, considering the condition one of hypertrophy with deposit of lime salts.

Schützemberger's case (81) is thought by Baumgarten to bear resemblance to the Ilg-Gruber case. The patient died in convulsions at forty years after having suffered from headaches, epileptiform attacks, and mental enfeeblement for some years. The skull was thickened and hard, diploë and sutures absent. The cavity was small, thickening being mainly at the expense of the inner table. The foramina were slightly diminished in size.

Huschke's case (64) was that of a woman seventeen years old whose skull weighed 3,224 grams and measured 228 mm. in length and 157 mm. in width. The thickness of the frontal bone was 10-35 mm., of parietal 22-35 mm., of occipital 16-40 mm. The diploë was mostly replaced by hard bone. The author considered the lesion due to rickets, a view which has been proven untenable by Virchow, Schmidt and others.

Gaddi (60) described a skull found in 1862. It weighed 3,660 grams. Surface rough, sutures obliterated. The bones of the face very large, the superior maxillæ particularly enormous. The lower jaw weighed 422 grams. The only other bone found, the second cervical vertebra, was also enlarged.

The first thorough sifting and elaboration of this accumulated material was made by Virchow, who also proposed the name *leontiasis ossea* (90), which was suggested to him by the leonine

appearance, in the case of Forcade in particular. He expressed the opinion that the process corresponds to elephantiasis of the soft parts, and that erysipelas plays an important part in the etiology. The latter assumption has not been borne out, though in a few cases the onset has been coincident with erysipelatous attacks, for instance, two of the cases of hyperostosis in epileptics related by Sommer (84). Virchow called attention to the fact that the hyperostotic bone was sometimes described as soft and spongy, but more commonly as hard and ivory-like, and showed that the latter condition is of two kinds, either an osteosclerosis which is secondary to the spongy condition or an eburnation which is primary. A concentric arrangement of the lamellæ indicates an osteosclerôsis, one parallel to the surface eburnation.

The case of Bickersteth, fully reported by Murchison (70), is probably to be considered one of leontiasis, although the hyoid bone and the fibula were also involved. At the age of fourteen years enlargement of the facial bones commenced, that of the fibula at twenty-seven. Death occurred at thirty-four, preceded by symptoms of compression of cranial contents, severe headaches, exophthalmos and blindness of the left eye. At the autopsy the superior maxillary bones were found enlarged, the inferior maxillæ enormously so. All bones of the cranial vault were involved except the occipital. The skull was covered externally with excrescences, the orbits, nasal and external auditory openings reduced in size. Histologic examination was confined to the fibula, "as it was not considered necessary to injure the cranium."

Wrany (93) reports a case where the disease commenced in the lower jaw, which is unusual. The skull was thickened and hard, the sutures obliterated internally. The frontal bone measured 12 mm., the parietal 6 mm., the occipital at the inion 20 mm. in thickness. The posterior wall of the sella turcica was atrophic and perforated. The inferior maxilla was thickened in all parts except the condyles, the superior maxillæ and lachrymal bones were also involved. No bone outside the skull was involved.

Le Dentu (56) reports a case of hyperostosis of the facial bones of two years' duration in an eighteen-year-old girl. A piece was excised and pronounced to be sarcomatous by competent men at the College de France. Le Dentu did not accept the diagnosis as healing took place after excision, and designates

the condition a "diffuse non-syphilitic periostosis." However, Baumgarten and Schmidt are inclined to consider the diagnosis of sarcoma correct. This controversy is of interest in connection with our own case, where we evidently have to deal both with leontiasis ossea and sarcoma.

Skull No. 205 in the Museum of the Breslau Pathologic Institute, described by Fischer (59) as an instance of leontiasis, is not mentioned in the treatises of Baumgarten (53) and Schmidt (82). The left side of the frontal and both parietal bones were involved. The tumor-like thickening of the frontal bone measured 36.5 cm. in circumference, 15 cm. in length, 6.5 cm. in height, 12.5 cm. in width. The bone was of ivory hardness, uneven, the inner surface covered with coarsely granular osteophytes. The cavity of the skull was very little narrowed. He also mentions the case of a man, eighteen years old, with a similar tumor the size of a fist at the right frontal eminence, which had existed for six years without any symptoms of compression. Fischer also describes the skull of a woman, who died of pulmonary tuberculosis, with bony tumors of the left temporal bone, sphenoid bone, and left side of the lower jaw.

In 1881, Paquet (72) presented before a surgical society a woman, twenty-two years old, on whose left upper jaw a tumor was first noticed at the age of three years. It had grown rapidly the last two years only, before that very slowly. The left eye was compressed and nasal fossa almost obliterated. The tumor was resected and histological examination showed most of it to be hyperostosis, but it was sarcomatous in the region of the antrum and palate. Baumgarten is of the opinion that two distinct affections were present, namely a hyperostosis, probably congenital, and a sarcoma of two years' standing.

The osteoporotic and much thickened parietal bone found in an Egyptian tomb, preserved in the Museum of the Royal College of Surgeons, London, and described by Eve (58), if an instance of leontiasis, is probably the oldest specimen of that disease in existence.

Silcock (83) reported the case of a man, who, at the age of twenty-three received a blow on the left forehead followed in five days by epileptic attacks and in a year by a bony tumor. A year later, when it had reached the size of a Tangerine orange,

trephining was performed, the bone found 35 mm. thick and beset with small exostoses, and the left frontal sinus obliterated. No histologic examination was made, a fact to be regretted, as it would be desirable to establish histologic points of differentiation, if possible, between such rapidly growing exostoses and the slowly advancing typical leontiasis.

In the same year (1890) appeared Poisson's (74) account of a patient with onset at sixteen of hyperostoses of the superior maxillæ and obliteration of nasal cavity and antrum. Histologic examination showed a rarefied condition of the bone. A trophic disturbance of neuropathic origin is considered as the basis of the trouble.

In 1892 F. Baumgarten published his thesis entitled, "*La leontiasis ossea*" (53), in which a thorough review of nearly all recorded cases is given, together with a discussion of the etiology, pathology, symptoms, and diagnosis of the affection. No new cases are reported, but further details given of a case previously reported by Pean (73), who had extirpated multiple tumors of the palate, upper and lower jaws in a woman, twenty-two years old. At the age of nine the first tumor had appeared in the right palatal vault. Histologically all the tumors presented the same structure. Baumgarten hesitates whether to call them fibro-sarcoma, osteo-fibroma, or osteo-fibro-sarcoma. The tumor of the lower jaw was surrounded by a fibrous capsule. There was no extension to the cranium. Baumgarten does not consider this a case of leontiasis. However, considering the long duration, it seems to the writer that this may be another instance of sarcoma arising in hyperostotic bone.

Starr (86) reported a case of leontiasis ossea or, as he proposed to call it, "*megalo-cephalie*," in a woman fifty-two years old, with onset at the unusually late age of forty-four. There was general hypertrophy of the bone over the entire calvarium, more marked just behind the temples. The head measured 24 inches in circumference. The cavities of the nose and mouth were encroached upon, the soft parts of the head and neck also thickened. Many nervous symptoms were present. Thyroid extract was tried without effect.

In 1895, Horsley (63), reported five cases of leontiasis ossea which he defined as "the development of bony new formation re-

sembling an overgrowth of diploë, and attacking the cranial bones (by preference the frontal), or more rarely the facial bones, especially the jaws." In a summary he says: "In all five cases the disease began in childhood or in early youth. In the three cases in which the disease attacked the eyebrow and roof of the orbit early, the displacement of the eyeball downwards occurred before any notable swelling appeared above in the supraorbital ridge. In the same three cases the region of the external frontal process was the focus of the malady. In no case was there any evidence of syphilis nor any really direct history of traumatism. In all cases the prominent symptom of distress was the pain produced by the entanglement and pressure upon the branches of the fifth nerve passing through, or by the affected bones. In the cases where operation was possible the mass was readily removed piecemeal, starting from the border of conjunction with the healthy bone."

Case 1. Male, age 19, onset at 15 with swelling above left brow, headache, vomiting, epileptic fits. Thyroid gland enlarged. Swelling of vertex, affecting frontal, right temporal, part of parietal and occipital bones. No operation. Case 2. Female, age 26, for five years swelling of left side of head. In 1888 the thickened bone was removed by operation. Recurrence, a second operation in 1894, with relief of all symptoms. Case 3. Male, age 28, onset at 20 with exostoses on each side of nose. Five years later protrusion of right eyeball and optic atrophy. Right superior and inferior maxillary bones were resected. Three years later operation on left side of face which had become involved. Case 4. Female, age 13, swelling of left side of frontal bone, resected. Case 5. Male, age 35, swelling of right side of frontal bone for at least 17 years. Resection of diseased portion of bone with relief of pain. Horsley gives the following anatomic description: "Outer surface of tumor in every case smooth, periosteum thickened. When the latter was stripped off the diseased bone presented a marked contrast to the normal, in being notably more vascular, the surface being extensively filled with minute foramina, the branching of superficial vessels very prominent, and the color a deep red as compared with that of normal bone. There was no demarkation between the tables and the diploë. Histologic examination was complete only in cases 2 and 5. The outer, rather harder portion of the bony mass consisted of

normal trabeculæ of bone separated by unusually wide medullary spaces. The lamellæ in the bone trabeculæ were well marked, but homogeneous throughout from the margins of the medullary spaces inward. In this region the medullary spaces were filled with a delicate, almost myxomatous connective tissue in the meshes of which hardly any characteristic medullary corpuscles appeared, except at the periphery of each space, where a complete row of osteoblasts was present in most cases. The central, softer and more friable portions of the tumor presented very different appearances from the foregoing. The medullary spaces were very large, the bony trabeculæ in process of active absorption and medullary tissue replaced by a neoplastic, fibrous growth. To consider these changes in further detail the alteration in the marrow may be taken most conveniently first in order. The medullary tissue of the outer portions of the tumor has already been stated to consist chiefly of a delicate fibrous tissue. From this condition to one in which the whole medulla was converted into a dense feltwork of fibrous reticular substance, every stage could be found. In the progress of this metamorphosis the individual fibers appeared to become thickened and to take a brownish stain with hematoxylin. While in the extreme stages the greatly thickened fibers assumed a more granular appearance, the whole suggested an osteogenic condition. Soon after the fibers became denser the osteoblasts disappeared and their place was taken by osteoclasts. The bony trabeculæ, as soon as the medullary tissue became obviously altered, showed notable changes, until in the most advanced areas of disease every bone trabecula exhibited by change in the staining reaction (deeper color) and morphological alteration of the cells, a marked reversion to a chondroid stage, and furthermore the margins and the surfaces of the trabeculæ displayed many false Howship's lacunæ." The extreme rarity of histologic descriptions renders the reproduction of this one in extenso justifiable. Horsley concludes by saying: "Leontiasis ossea is a pathologic entity in which both osteoplastic and osteoclastic processes are represented, and that it is a hopeless condition, for which operation, if extensively practiced, offers considerable relief."

In 1896 a valuable article, entitled "Hyperostosis cranii," by Putnam (76), appeared in this country, with four new cases,

statistics of fourteen typical cases, and a brief critical review of the literature. Case 1. Female. Mother and sister had "suffered with their heads." At three, some disease of ankle, followed by separation of pieces of bone. Bright in youth but inclined to drowsiness. Onset at 19 with headache, tinnitus, later deafness, head gradually broadening. For years a purulent discharge from ears. Bilateral facial paralysis before death, also dimness of vision in one eye. Died at about 30. Autopsy showed a general purulent periostitis cranii. Diffuse thickening of cranium, most marked anteriorly, 1.5 cm. Bones with a worm-eaten appearance from caries. Weight of skull nearly twice the normal. Orbits and sella turcica smaller than normal. Grooves for vessels practically absent. Diploë ivory-like. Case 2. Male. Two bean-sized lumps noticed at the top of the head at five. They finally reached the size of $2\frac{1}{2}$ inches in diameter and $\frac{1}{4}$ inch in height. Prominent occipital protuberance and temporal ridges. Slight optic neuritis, eyes prominent. Epileptiform convulsions began at seven; deafness and dementia before death, at 22. No autopsy. Case 3. Man, observed at 50 years, had noticed that his head was growing larger for ten or twelve years, enlargement attended by neuralgic pains, impaired hearing, insomnia. Case 4. Female, age 35. Enlargement of head first noted in the region of left parietal eminence at 23. Burning sensations and numbness of face. Circumference of head 65.5 cm. Surface of cranial bones irregular from presence of exostoses. A case observed by Dr. Prince is also reported by Putnam in the same article. Male, struck on the forehead by a pulley, two years after noticed swelling of frontal bone, followed by symptoms of compression and death six years after the injury. The autopsy showed marked thickening of the skull in the mid-frontal region, of the temporal ridges, and particularly of the orbital plates.

Following Putnam's article is a report by Edes (57) of a woman, age 52, whose head had been growing larger for some years. Six years later (1902) this case was found by Prince (75) to have developed into one of *ostitis deformans*, almost all of the long bones in the body having become affected. There was also a general myasthenia.

White's case (92) was that of a man, 32 years old, with a prominent bony ridge running horizontally round the back of the

head, reaching within an inch of outer canthi of both eyes. This had appeared a short time after a fall from a window at the age of three years. Later in childhood he had fractured the left femur, at 22 the right femur, at 29 the right shoulder became stiff. The maximum circumference of the head was $24\frac{1}{2}$ inches. Facial bones normal. Slight nystagmus and bilateral deafness; no headache. General health good. According to Prince (*loc. cit.*, p. 810), this case later came to autopsy, recorded by Collier. Unexpectedly a condition of syringomyelia was found which had not given rise to the usual sensory symptoms. Prince thinks the bony changes were very likely of the nature of the trophic lesions dependent on that disease.

Noble (71) reported a case of a young man with extreme bulging of the nose and lower part of the forehead, the eyes being five inches apart. The disease had commenced with the appearance of a bony growth in the upper part of the right nostril, four months after the injury of the nose by a fall at the age of eighteen.

Young's patient (94), a man who died at 46, had prominent superior maxillary bones and supraorbital ridges with obliteration of the antra and frontal sinuses, and narrowing of the right orbit and both nasal cavities. Cranial bones of normal thickness, sella turcica normal.

Hinsdale's (62) patient, a woman, died at 71. There was diffuse hyperostosis of the skull, thickness varying from 14 mm. in temporal, to 35 mm. in occipital bone; circumference of head 71 cm. Diploë reduced in amount, bone unusually soft and friable. The skull-cap weighed 1870 grams. The lower half of the head was not enlarged. The epiphyses of both femurs were enlarged and covered with friable exostoses, and the long bones of the lower limbs were slightly bowed. Acromegaly or myxedema were excluded. The enlargement of the head was first noticed at sixty-four.

Sommer (84) contributes an article on cranial hyperostoses in the insane. Epileptics are particularly frequently affected. The anterior half of the base, especially the sphenoid, is most frequently involved. Twenty cases were studied.

Stack's case (85) was that of a woman who, at the age of three years, injured the forehead by a fall. The wound sup-

purated. Enlargement of the head was first noticed at the age of seven. She died at 21. At the autopsy the skull was found greatly thickened, the half skull without the lower jaw weighing $7\frac{1}{2}$ lbs. The thickening was greatest in the frontal bone. The bone was very soft. The lower jaw weighed 1 lb., and is described as follows: "The whole of the horizontal ramus was enlarged and rounded; the compact shell was expanded and filled with very porous bone, containing a quantity of very slimy, gummy material, which poured out when cut into." The nasopharynx contained a firm white tumor attached to the hard and soft palate, found on histologic examination to be a fibro-sarcoma with numerous myeloid cells. Brain and hypophysis normal. There were scoliosis, a soft and distorted pelvis, bent femora and tibiae, changes which led Prince to suspect that the case was a combination of hyperostosis and osteomalacia.

Schiller (80) reports a case from Czerny's clinic. Male, age 30. A brother was delivered by embryotomy on account of abnormal size of thorax. Patient delivered by forceps as head was unduly large. Abnormal increase in size of head continued until age of 19 years, when it became stationary. The only symptom was frequent headache. The circumference of the head was 61 cm. The head was weighed, hanging over the edge of the table, when the patient was anesthetized. Its weight was between 5 and 5.5 kilograms, while that of normal heads in the same position was 2 to 2.5 kilograms. There were prominences of the frontal and parietal bones, seen in X-ray pictures to be due to actual bony thickening. Facial and other bones not involved.

In the appended table a summary of the causes just recorded is given. Although they undoubtedly do not constitute a clinical entity, in our present state of knowledge it seems convenient to consider them together as all more or less possess the characteristics of Virchow's leontiasis ossea. In many of them, such as some of Horsley's cases, there may be only a resemblance to leontiasis, in others the latter may be a part of a more general disease, but as the nature of leontiasis itself, as well as of nearly all of the allied disorders, is unknown, only arbitrary distinctions can be made, when the phenomena overlap. Hence, we will consider these cases together, first as to their

Etiology—Heredity seems to play no part. The sexes are about equally affected. Onset nearly always in childhood or early youth, even before birth, if we include Breschet's and Schiller's cases. Of the five cases with onset after twenty-five, those of Starr, Edes, and Hinsdale were complicated by disease of other bones, and possibly belong to *ostitis deformans*. Trauma is said to have preceded the onset six times. Of these cases Buhl's was complicated by gigantism, Stack's by *osteomalacia*, White's probably was due to *syringomyelia*, Noble's and Silcock's were of such rapid course as to suggest a true tumor. The *erysipelas* and *suppurative inflammations* present in a few cases were evidently merely complicating or accidental. Baumgarten's view that the disorder is trophic and developmental is probably the best at present.

Morbid Anatomy and Histology—Sternberg (87) divides the cases into *diffuse hyperostoses* with involvement, rarely even, of all cranial and facial bones (cases of Malpighi, Jadelot, Sandifort, Ilg-Gruber, Gaddi), and localized *tumor-like hyperostoses*, such as the case Forcade, and those of Buhl and Bickersteth-Murchison. Such a line, however, seems rather artificial. As to the bones involved, the following may be learned from 30 fairly completely reported cases in our table:

Cranial, upper facial and lower jaw.....	10
Cranial only (or at least principally).....	10
Cranial and upper facial.....	4
Cranial and lower jaw.....	2
Upper and lower jaws.....	3
Upper jaw only.....	1

Of the cranial bones the anterior part of the frontal is usually most affected. Of 22 cases the disease is stated to have commenced in the cranial bones 11 times, upper facial 10 times, and lower jaw once (Wrany's case). The affected cranial bones are generally harder than normal, with absence of *diploë*, and obliterated sutures. Exceptions are the cases of Stack, Hinsdale, Buhl, and our own, where the hyperostotic cranial bones were abnormally soft. The weight of the skull is often enormously increased, as well as the thickness of the vault, as the frontal bone in our case, 85 mm. The external surface is generally rough, often with

large exostoses, while the internal surface is most commonly smooth. Narrowing of the cranial cavity, orbits, nasal cavities, sinuses, and foramina for nerves and vessels is common. The affected facial bones are usually the seat of nodular exostoses, diffuse thickening being much rarer than in the skull. Histologic examinations are too few to admit of any generalizations. The association with sarcoma in as many as five cases, those of Paquet, Stack, Pean-Baumgarten, Le Dentu, and ours, must be more than a coincidence. The descriptions in these cases lead us to infer that the sarcomatous process started in the periosteum of the diseased bone. In only one of these cases, that of Le Dentu, is it possible that the entire process in the bone was due to sarcoma. One may also suspect that some of the more acute cases following injuries, as those of Silcock and Noble, really were instances of osteosarcoma. The complications with gigantism (cases of Buhl, Sirena (42) and ours), with syringomyelia (White), osteomalacia (Stack), ostitis deformans (Edes), and acromegaly (Saucerrotte), and possibly a case of Sternberg's (87), are extremely interesting and suggest an etiologic relationship between all these obscure conditions.

Symptoms—Most of these are explained by pressure on the brain or nerves. Headache is the rule. Anosmia, blindness, ocular paralysis, neuralgic pains, numb or anesthetic areas, tinnitus, deafness, dysphagia, speech troubles, epileptiform convulsions, mental disorders, have all been recorded. Optic neuritis was recorded in Putnam's case, that of Edes, and ours. The course is very slow, but nearly always progressive, usually extending over ten to thirty years.

Diagnosis—*Ostitis deformans* usually begins after forty, and the long bones are here involved. When it begins in the cranium, a differential diagnosis is extremely difficult or impossible until the long bones become involved. Recently Prince (75) has advocated the view that the two perhaps are only different manifestations of the same disease, and probably trophic disorders, that the underlying cause may be found in the nervous system which he makes a plea to examine more closely in the future. *Acromegaly*.—The enlargement of the hands, feet, tongue, and other soft parts, makes a distinction easy in a pronounced case, but, as stated, here we also have combinations of the two. *Sarcoma*.—Diagnosis may

be very difficult when the maxillary bones are affected, particularly in the case of multiple, slowly growing tumors. Slow growth and gradual enlargement of the skull are in favor of hyperostosis. The tendency for sarcoma to arise from hyperostotic bone must be borne in mind.

Prognosis—Unfavorable, as the tendency is for the encroachment on cavities and foramina to progress steadily.

Treatment—Very little is to be expected until the underlying morbid tendency can be directly antagonized. Operation may afford relief or even cure, if performed early, but in most cases the disease is too diffuse for surgical interference, and the reappearance of the disease at some other point is likely when excision of the primary focus may be possible. This indeed is to be expected if we are dealing with a developmental trophic disorder.

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OBSERVER	SEX	AGE AT ONSET	DEATH AGE AT	BONES OF SKULL AFFECTED	OTHER BONES AFFECTED	PART FIRST AFFECTED	CONDITION OF DISEASED BONE	CONDITION OF CRANIAL SUTURES	SYMPTOMS OF COMPRESSION OF BRAIN OR NERVES	REMARKS
1. Malpighi	?	?	?	All cranial parts. U- larly temporal, tumor of cher- ry size near sagittal sut- ure. Orbits small.	?	?	?	Effaced.	?	Skull weighed 3618 grams. Nothing else observed.
2. Jourdain, Ribet (Case For- cade).	M	12 yrs.	45 yrs.	Upper lower nasal, all cranial bones. Orbits small.	All unusual- ly delicate.	Right upper jaw.	Covered with large, hard ex- ostoses.	—	Blindness. Speech trou- bles.	Onset with ab- scess at inner canthus. Dried skull weighed 4,192 grams. Lower jaw alone 2,792 g. Facial muscles fibrous and ad- herent to peri- osteum.
3. Sandifort.	?	?	?	All cranial.	?	?	Skull thickened by one inch. Abundant dip- loe. Exostoses on inner sur- face, particular- ly of frontal bone.	—	?	Skull only ob- served.

4. Jadelot.	?	?	?	All cranial.	?	?	Thickness 19.8 to 40.2 mm. Very compact.	—	?	Skull weighed 3,913 grams.
5. Saucerrotte.	M	?	39 yrs.	Cranial and lower jaw.	?	Spine, ribs, sternum, clavicles. Bones of extremities.	?	—	?	Lips were thick. Circumference of head 797 mm. Probably acromegalic.
6. Ilg-Grubert.	F	10 yrs.	27 yrs.	All cranial, most so occipital. Sella turcica shallow. All foramina small. All cranial.	?	?	?	Mostly disappeared.	Blindness, deafness, dysphagia, anosmia, headache, delirium, epileptic fits.	Skull without lower jaw, teeth and nasal bones weighed 4,200 g.
7. Kilian.	?						Thickness 27-54.1 mm., latter parietal. Compact. No diploe. Thickness 27-67 mm.	—		
8. Bojanus.								Mostly disappeared.		Skull without lower jaw weighed 4,000 g.
9. Breschet.		Before birth.	18 mos.	All cranial, facial not involved.	Probably none.	?	Greatest thickness of vault 1 inch. Ant. fontanelle present. Thickness 7-20 mm.	?	Convulsions since birth.	
10. Vrolick.	?	?	?	All cranial. Sella turcica narrow and deep. All cranial. Foramina small.	?	?	?	Obliterated except squamous.	?	Skull weighed 1,305 grams.
11. Schützemberger.		?	40 yrs.				Hard, thickened at expense of inner table. No diploe.	Obliterated.	Convulsions, headache. Loss of speech. Mental enfeeblement.	

OBSERVER	SEX	AGE AT ONSET	AGE AT DEATH	BONES OF SKULL AFFECTED	OTHER BONES AFFECTED	PART FIRST AFFECTED	CONDITION OF DISEASED BONE	CONDITION OF CRANIAL SUTURES	SYMPTOMS OF COMPRESSION OF BRAIN OR NERVES	REMARKS
12. Huschke.	F	?	17 yrs.	All cranial.	?	?	Thickness 1-4 cm. latter oc- cipital Hard. little diploe. Surface rough.			Skull weighed 3,224 grams.
13. Gaddi.	?	?	?	Facial and cra- nial.	Axis en- larged.	—		Obliterated.	?	Skull weighed 3,660 g., lower jaw 422g. Skull and axis only bones examin- ed.
14. Bicker- steth. Murchison.	M	14 yrs.	34 yrs.	Cranial except occipital. Up- per and lower jaws.	Hyoid and fibula.	Facial bones.	Skull covered with excres- cences.		Headache, blindness.	
15. Wrany.				Cranial and fa- cial.	None.	Lower jaw.	Hard. Thick- ness of cranial 6-20 mm.	Obliterated internally.	Insanity.	Preceded by trauma.
16. Buhl.	M	9 yrs.	25 yrs.	Facial and cra- nial. Left or- bit small.	Scoliosis.	Facial bones?	Frontal 6 cm. thick, soft. Many exostos- es on facial bones.		Death ascribed to cerebral compression.	Gigantism, hgt. 235 cm. Onset after kick on cheek.
17. Le Dentu	F	16 yrs.	?	Facial.	None.	Facial bones.		?	None.	Operation. Probably sar- comatous. Ob- served at 18.

18. Fischer.	?	?	?	Frontal and parietal.	?	?	Uneven, hard. Large exostosis of frontal bone.	?	?
19. Fischer.	M	12 yrs.	?	Frontal bone.	None.	Frontal bone.	Tumor size of fist at right frontal eminence.	?	Observed at age of 18.
20. Paquet.	F	3 yrs.	?	Left upper jaw.	Probably none.	Left upper jaw.	Historically partly hyperostosis, partly sarcoma.	?	Observed at 22. Growth rapid last two years. Operation.
21. Silcock.	M	24 yrs.	?	Left frontal.	None.	Left frontal.	Tumor of frontal 35 mm. thick. Small exostoses on inner surface. Frontal sinus obliterated.	?	Observed at 25. Onset one year after blow on head. Operation.
22. Poisson.		16 yrs.		Upper jaw.	Probably none.	Upper jaw.	Hyperostosis of superior maxillary bone. Obliteration of nasal cavity and max. sinus. Multiple tumors.	?	Histologic exam. showed a rarefying osteitis.
23. Pean, F. Baumgarten.	F	9 yrs.	?	Hard palate, upper and lower jaw.	None.	Hard palate.		?	Histologically, between fibrosarcoma, osteofibroma and osteofibrosarcoma. Observed and operated at 22.

OBSERVER	SEX	AGE AT ONSET	AGE AT DEATH	BONES OF SKULL AFFECTED	OTHER BONES AFFECTED	PART FIRST AFFECTED	CONDITION OF DISEASED BONE	CONDITION OF CRANIAL SUTURES	SYMPTOMS OF COMPRESSION OF BRAIN OR NERVES	REMARKS
24. Starr.	F	44 yrs.	?	Calvarium and facial bones. Nasal and oral cavities small- er.	Cervical vertebrae				Nervousness, irritability, numbness of fingers, uncer- tain move- ments.	Observed at 52. Circumference of head 24 in. Skin thickened.
25. Horsley.	M	15 yrs.	?	Entire vertex.	None.	Frontal bone left side.	Smooth exter- nally with min- ute foramina. More vascular than surround- ing bone, no demarkation between tables and Periosteum thickened.	?	Epileptic fits, and vomiting.	Observed at 19.
26. Horsley.	F	21 yrs.	?	Left side of head		Frontal bone left side.			Pain.	Operation twice. Observ- ed at 26.
27. Horsley.	M	20 yrs.	?	Nasal, superior and inferior max. bones. Frontal bone.		Nasal bones.			Pain.	Operation at 25. Recurrence; 2d operation at 28.
28. Horsley.	F		?			Frontal bone left side.			Pain.	Operation at 13.
29. Horsley.	M	18 yrs.	?	Frontal bone.		Frontal bone right side.			Pain.	Operation at 35.

30. Putnam.	F	19 yrs.	28 yrs.	Cranial malar bones. Orbits smaller. Sella turcica narrow.	None.	Cranium.	Diffuse thickening of cranium, most marked ant. Extensive caries. Diploe ivory-like. Two broad exostoses on top of head. Occipital protuberance and temporal ridge prominent.	Obliterated.	Headache, pain in ears and eyes. Deafness. Facial paralysis.	Suppurative periostitis cranii. Weight of skull nearly twice the normal.
31. Putnam.	M	5 yrs.	22 yrs.	Frontal and parietal.	None.	Frontal and parietal.		?	Epileptic fits. Deafness. Optic neuritis. Dementia.	No autopsy.
32. Putnam.	M	38 yrs.	?	Cranial bones. Edges of orbits thickened. Facial bones uninvolved. Cranial bones.	None.	Cranial bones.		?	Neuralgic pains. Throbbing sensation in rt. ear. Impaired hearing. Burning sensation in head, numbness of face. Sometimes diplopia.	Abscess of both ears at 35. Observed at 50.
33. Putnam.	F	23 yrs.			None.	Left parietal bone.	Calvarium covered with exostoses.	?		Observed at 35. Circumference of head 65.5 cm.
34. Prince. Putnam.	M	Adult.		Frontal bone.		Frontal bone.	Frontal bone thick, particularly orbital plates. Temporal ridge prominent anteriorly.	?	Onset two yrs. after injury to frontal bone. Mental dulness. Vomiting. Rt. optic atrophy.	Death 4 yrs. after onset.
35. Edes. Prince.	F	40-50 yrs.		Cranial bones, mainly frontal.	Nearly all long bones.	Cranial bones.	Spine and long bones deformed.	?	Weakness of gait. Deafness. Rt. eye nearly blind.	First observed at 52. Probably otitis deformans.

OBSERVER	SEX	AGE AT ONSET	AGE AT DEATH	BONES OF SKULL AFFECTED	OTHER BONES AFFECTED	PART FIRST AFFECTED	CONDITION OF DISEASED BONE	CONDITION OF CRANIAL SUTURES	SYMPTOMS OF COMPRESSION OF BRAIN OR NERVES	REMARKS
36. White. Collier. Prince.	M	3 yrs.	Abt 35 yrs.	Cranial only.	None.	Cranial bones.	Projecting ridge around back of skull.	?	Bilateral deaf- ness. Slight nystagmus. Fits. Spastic atro- phic paralysis of arm.	Onset follow- ing injury. Op- eration for fits. Autopsy show- ed syringomy- elia. Onset 4 mos. after fall on nose.
37. Noble.	M	18 yrs.	21 yrs.	Nasal, frontal, sup. max. bones.	None.	Right nasal bone	Extreme bulg- ing of lower part of head.	?	Three attacks of insanity.	Bosses of sup. max. bone found cancel- lous on section. Marked athe- roma of aorta.
38. Young.	M	39 yrs.	46 yrs.	Sup. max. and frontal bones. Rt. orbit and both nasal cav- ities diminish- ed.	None.	Left sup. maxillary.	Symmetrical protrinent bos- ses of sup. max. and fron- tal bones. Vault of norm- al thickness. Frontal sinus- es and antra occluded.	?	Anosmia. Dim- inution of vision.	
39. Hinsdale	F	64 yrs.	71 yrs.	All cranial.	Slight bow- ing of the long bones of extrem. Exostoses of femurs.	?	Extremely soft. Diffuse hyper- ostoses of cra- anium. Thick- ness of vault 14-35 mm.			Skull cap weighed 1,870 gms., brain 1, 360 gms. Cir- cumference of head 71 cm.

40. Stack.	F	7 yrs.	21 yrs.	All cranial. Lower jaw. Palate.	Scoliosis. Soft and distorted pelvis, and tibiae bent and soft.	Cranium.	Bones soft. Fibro-sarcoma of naso-pharynx.	The half skull without lower jaw weighed 7½ lbs., lower jaw 1 lb. Injured forehead at 3. Probably complicated by osteomalacia.
41. Schiller.	M	Before birth. ?		Frontal, parietal and occipital bones.	None.	?	Tumor - like prominences of frontal and parietal bones. Prominent ext. occipit. prot.	Observed at 30. Circumference of head 61 cm. Birth difficult on account of size of head.
42. Present Case.	M	9 yrs.	28 yrs.	All cranial. Left facial.	Scoliosis.	L. frontal.	Cranial bones soft, thick, invaded by sarcoma. Frontal 85 mm. thick.	Gigantism, hgt. 245 cm.
							Prominent.	Headache, blindness, immobility of eyeballs. Ptosis. Facial anaesthesia. Deafness, tinnitus.

STUDIES UPON THE CEREBRAL CORTEX IN THE NORMAL
HUMAN BRAIN AND IN DEMENTIA PARALYTICA.

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(Continued from page 554.)

Technique. Turning to the technique employed in this work, first will be described a method used in transporting material con-

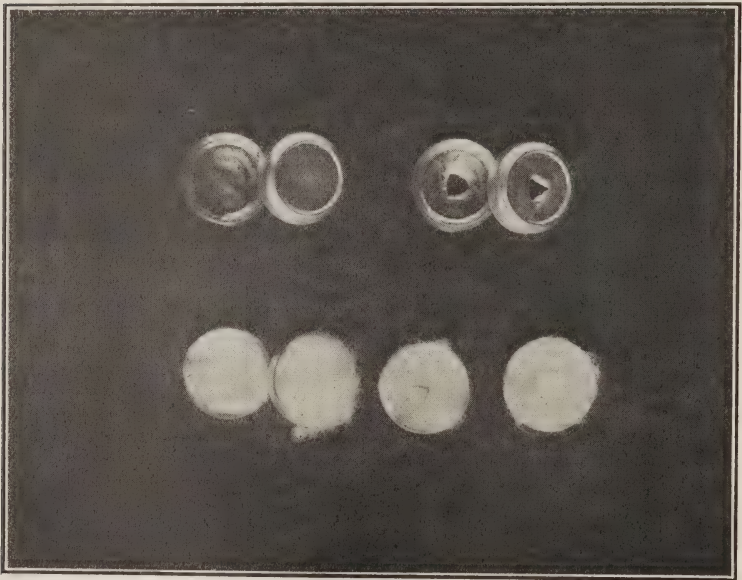


Fig. A.

siderable distances in the minimum amount of space, and preparing the same in the shortest possible time.

On several occasions the writer was enabled to secure more than one brain at the same time, and at a considerable distance from the laboratory. It was desired to place small blocks from various parts of each of the brains in various fixative media in the shortest possible time, and to put them into the smallest possible

space for transportation. The smallest size of tin boxes known as "Miller's patent seamless box" (Fig. A) were secured at trifling cost at Eimer & Amend's, in New York city; though any small box would answer the purpose. These measured but 2.5 c. c. in diameter and 1.25 c. c. in depth. One gross of these boxes occupy only a space of 15 c. c. square by 5 c. c. in depth. By piercing a

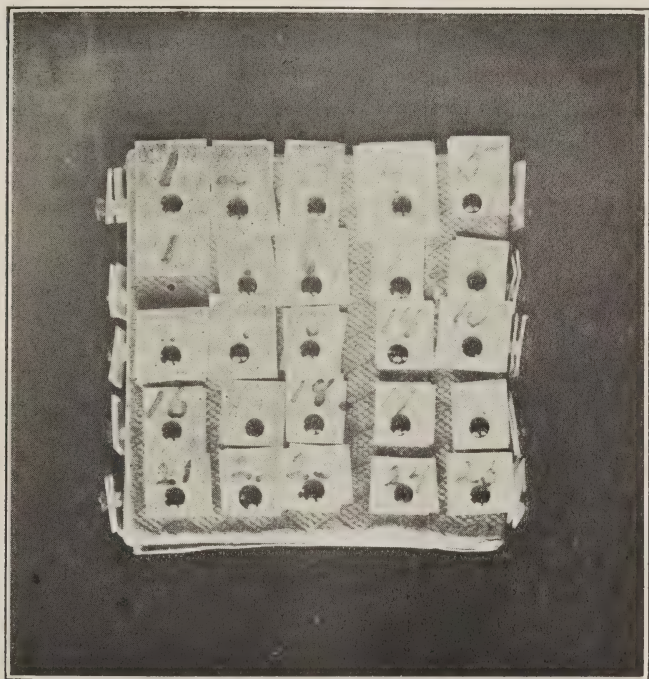


Fig. B.

hole in both the cover and bottom of these boxes a free circulation of the fixative medium is secured—as mentioned, any other small box or phial admitting of the free circulation of the fixative medium could be used; but these chanced to be the most convenient and available receptacles to the writer. By placing a bit of absorbent cotton or cheesecloth in both the bottom of the box and in the cover the specimen is perfectly protected. A square box of black pins (Fig. B), such as can be secured at any drygoods store at trifling cost, was purchased. Small squares of cardboard

were cut out and numbered in duplicate, using a lead pencil. Each number, with its single duplicate, was placed upon a pin and returned to its position in the box. The various fixative fluids desired were carried in small glass jars with ground glass covers. All these preparations are made in advance so that at the autopsy, upon removing the brain, small pieces can be taken from any part of the cortex, each one placed in a separate small tin box with a number from one of the pins. The other or duplicate number is left upon the pin, and the latter thrust into the space on the brain surface from which the block was taken (Plate I, Fig. 1). The small tin box is then thrown into whatever fixative fluid is desired, and the fluid enters and fills up the interior by means of the openings above described, the whole process taking less time than required in explanation. In this way a large number of blocks can be taken from one or more brains without any possibility of confusion, and in a minimum amount of time, and likewise take up a minimum amount of space, requiring no writing or labeling of specimens at the time. After all blocks desired are removed the brain is carefully placed upon cotton in a tin pail of the required size in whatever fixative fluid is desired, and other brains can be treated similarly.

At the laboratory subsequently the exact locality from which blocks were taken can be noted at leisure in the most accurate manner. The writer has by this method taken blocks from various parts of two separate brains, placed them in half a dozen different fixative fluids, and packed them up, together with the two brains in tin pails of suitable diameter, placing everything in a hand bag of medium size, and carried the same many miles on the train without the slightest inconvenience or knowledge by others of the contents of the hand-bag. The fixative agents employed in this work were alcohol absolute, alcohol 95 per cent, formalin 10 per cent solution (40 per cent formaldehyde, 1 part by volume, water 9 parts by volume), Van Gehuchten's fluid (alcohol absolute 60, chloroform 30, and glacial acetic acid 10 parts by volume), and Lang's solution (mercuric chloride 5 grams, sodium chloride 6 grams, acetic acid 5 grams, and water 100 grams). The most satisfactory results have come from fixation in Van Gehuchten's fluid, and the alcohols. In the employment of the latter more or less shrinkage results, but this can be

recognized, and the chromatic substance within the cell is usually well marked. Van Gehuchten's fluid was used as follows: Small blocks, not more than .3 to .5 c. c. in thickness, were immersed in Van Gehuchten's fluid and left for twelve hours. They were then placed in 95 per cent alcohol, where they remained until desired for use. No changes in the contour of the cells were observed to result, and the chromatic substance in these cells was well shown in the subsequent staining. Blocks to be imbedded were then placed in absolute alcohol, and, for the paraffin method, transferred to xylol, and left in the latter for several hours till thoroughly permeated. They were then placed in paraffin—melting point about 50° C., for from 45 minutes to one hour, and then transferred to another paraffin bath of the same melting point for the same length of time, so as to secure complete penetration and the removal of all xylol. The blocks were then imbedded and subsequently sectioned by a Minot microtome serially. The sections were made varying in thickness from 2 to 15 microns, those from 6 to 10 microns being found best for study of the arrangement and internal structure of the cells. The celloidin method was also employed, but it was more difficult to secure thin sections when desired, and especially to arrange them serially. A thin smear of egg albumin was placed upon a perfectly clean slide, several consecutive sections placed upon the same, and then a small quantity of water by means of a pipette allowed to flow under the sections. This was gently warmed upon the water bath or over an alcohol lamp until the sections were perfectly flattened out. The water was drawn off and the sections allowed to dry. This treatment permitted all subsequent manipulations with the sections upon the slides, without their floating off or becoming disturbed or injured. The slides were then placed in xylol to dissolve out all paraffin and run down in successive grades of alcohol from absolute alcohol to 30 per cent alcohol, and from the latter immersed in water. The sections were then stained upon the slide by means of an aqueous solution of methylene blue, as given by Nissl (methylene blue, 3.75 grams; Venetian soap, 1.75 grams, and distilled water 1,000 c. c.), or by a 1 per cent aqueous solution of methylene violet or a 1 per cent aqueous solution of thionin. A counter stain of erythrosin was used in some cases after treatment with methylene blue, giving the achromatic substance a pink color in

contrast to the blue color of the chromatic substance. The routine method was to gently heat the slide covered with the stain for two minutes over an alcohol lamp, keeping the slide in constant motion, and only allowing it to become sufficiently heated so that the steam would come from the surface, but no bubbling of the dye in solution. The dye was then gently washed off, the slide immersed in water, and then run up into 30, 50, 70, 80, 95 per cent, alcohol, absolute alcohol plus xylol equal parts, xylol, and finally mounted in xylol-damar. Some slides were placed, after immersion in water following the staining process, in anilin-oil 10 parts, absolute alcohol 90 parts, and subsequently treated as described by Nissl, and finally mounted in benzene-colophonium. The first method, however, gave the most satisfactory results in the hands of the writer, and which, as seen, embodies various modifications of the original Nissl method. Sections prepared in the routine method above described have been frequently examined under the microscope, also frequently exposed to sunlight, and at times to the powerful rays of the electric arc in the photomicrographic work, but were in nowise faded or injured after a period of two years from the time of preparation.

In the micrographic work the most painstaking care was employed in every detail of the work. Achromatic objectives of various power with compensating eye-pieces in different combinations were used so as to produce different magnifications up to 1,400 diameters. Oil-immersion lenses were not used, as the above combination secured the greatest possible depth under such high powers. The time of exposure varied from a few seconds in the low power photomicrographs to five minutes in some of those magnified 1,400 diameters.

Material. Some twenty brains were secured, and sections made from various parts of them all and studied in connection with this article. Three brains of cases electrocuted at Ossining, and in which the autopsy occurred immediately afterwards and the material placed in various fixative media, were secured. The photomicrographs representing the practically normal brain histologically in the first part of this article were taken from one of these brains, marked A (Plate I, Fig. 1), a man thirty-six years of age who had been confined in Sing-Sing State Prison for over two years and leading the regular routine prison life during that

period. The autopsy was held immediately after death by electrocution. The body was well nourished, and no pathological condition of the central nervous system or other organs was found. Small blocks, .3 to .5 c. c. in thickness, from various convolutions of the brain were placed in the various hardening or fixative agents described above, and the brain itself immersed in 95 per cent alcohol. The blocks were subsequently imbedded in paraffin,

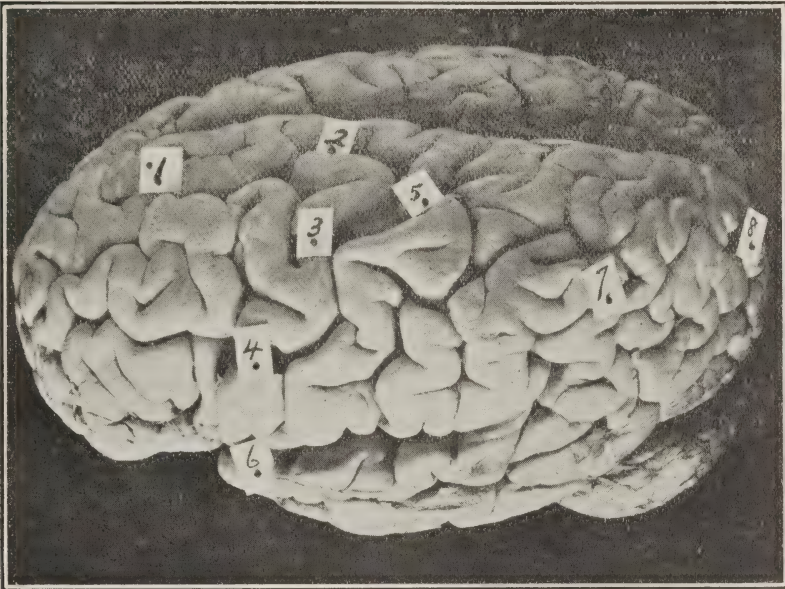


Plate I, Fig. 1.

sectioned serially, stained, differentiated, and mounted as already described.

Brains from three cases of dementia paralytica were also secured and studied. Sections from the one marked B (Plate X, Fig. 29) were used to illustrate this article in its pathological portion. The paralytic dement from which brain B was taken was a lawyer, had a collegiate education, was single, no history of syphilis, and family history negative. Was native of the United States. The disease began at about the age of twenty-eight years with the usual change of character, followed by grandiose ideas

and maniacal excitement. Asylum treatment for over a year caused his symptoms to clear up sufficiently so that he was discharged. For nearly two years he was in a fairly quiet condition. He then broke out in a long period of excitement, lasting over a year, followed by terminal dementia lasting for one and a half years before death, which latter was uncomplicated. Duration of the disease was thus over five and a half years, during which period there was a remission lasting two years, in which he did not require asylum treatment. There was contracture of both arms and legs for three months preceding death. Mentally he was a little brighter during that time. At death the body was immediately placed upon ice, and the autopsy performed fifteen hours later. The body weighed 78 pounds at death and was flexed. Two small cavities, each the size of a pea, were found in the apex of the right lung. The lymphatics were somewhat enlarged. Heart was atrophied. There was meningeal thickening. The ependyma in the posterior part of the floor of the fourth ventricle was very slightly granular. The membranes were anemic, and there was no edema or fluid. The convolutions, especially in the region of the central convolutions, are widely separated from one another; but, as will be described later, this is largely due to mechanical causes in subsequent fixation. In addition to these brains over a dozen were secured from other sources, mostly from the New York City Morgue, of persons who had committed suicide or died suddenly from accident or homicide, with autopsy performed within a few hours after death.

Normal Cortex. The descriptions and plates herein contained, and illustrating the normal human cortex, are not intended to give the idea of being in any way a complete exposition of such a vast subject; but it has been the aim to give photomicrographs with accompanying descriptions of typical sections and cells from various regions of the cortex, as a basis of comparison with sections taken from corresponding regions of the cortex in a case of dementia paralytica. Neither is this contribution intended to be in the nature of an atlas, being much too limited in scope for such a work; but to illustrate as far as practicable the structures found in what can be considered a fairly normal brain, with the conditions found in a brain of one dying from dementia paralytica. Various regions of the normal cortex from brain A, one of the

electrocuted cases (Plate I, Fig. 1), with its accompanying plates will be first described, and then will follow a like description of the corresponding region with its accompanying illustrations of the brain of the case of dementia paralytica, brain B (Plate X, Fig. 29). Plate I, Fig. 1, above referred to, shows the left hemisphere of Brain A, natural size; a normal brain, with well marked convolutions and sulci. The small pieces of cardboard with their

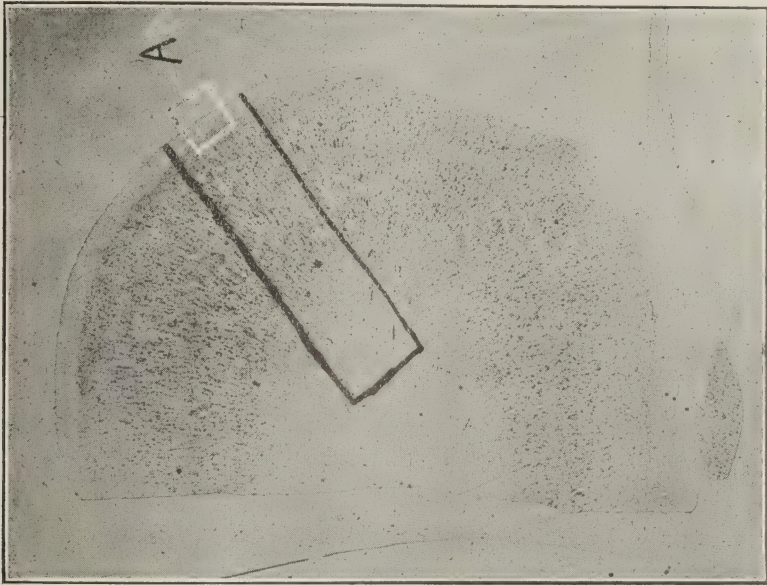


Plate I, Fig. 2.

contained figures show the particular point from which the subsequent blocks were taken, and also illustrate the method of exact localization of blocks described under the heading of technique.

Frontal Region.—Turning now to the several cortical regions, various portions of the first, second and third frontal convolutions were examined in different brains, and typical of this region is the section shown in Plate I, Fig. 2, which, with minor modifications, is similar to that of all portions of this region, and will now be described in detail. The block from which this section was taken comprised a part of the external surface of the first, or superior frontal convolution, including the entire width of the gyrus for a distance of .4 cm., and taken from the

position shown by the figure 1, Plate I, Fig. 1. The gyrus at this point measures .6 cm. in width, so that the actual size of this section is .6 cm. in width by .4 cm. in depth, and it is 6-2-3 microns in thickness.

The block at autopsy immediately after death was placed in Van Gehuchten's fluid, where it remained for twelve hours, when



it was placed in alcohol 95 per cent until ready for use. It was then imbedded in paraffin, sectioned serially with a Minot microtome, stained for two minutes in warm methylene blue, decolorized in alcohol, cleared in xylol, and mounted in xylol-damar. Plate I, Fig. 2, referred to above, shows the section under a magnification of 14 diameters. The section has been broken in manipulation, but shows the general striated arrangement of the cells radiating outward from the white medullary center to the surface; also the layering of the cortex. The strip of cor-

tex outlined in ink on the plate at *a* is that portion shown in Plate II, Fig. 3, under a magnification of 100 diameters. The outer pale layer, the thicker cellular layers, and the inner medullary white portion can easily be differentiated even with this magnification.

The outer layer is found to be fairly uniform in thickness in this section, but we will find later that there may be a considerable variation, not only in the thickness of this layer, but also of the other layers, and consequently of the entire cortex. The radial arrangement of the cells from the medullary white substance is especially well shown under this magnification, and a more general and comprehensive view of both the normal arrangements and of derangements and disturbances due to pathological changes can be made out under this magnification than by higher powers in which but minute areas are seen. These latter high magnifications are of course also necessary for the complete study of all the details of such variations or pathological changes. Plate II, Fig. 3, magnified 100 diameters, is that portion of the section outlined in ink in Plate I, Fig. 2 at *a*. Decolorization has been carried on here to such a degree as to make many of the cells appear somewhat pale and washed out, and to cause a complete decolorization of many of the neuroglia cells. This, however, admits of a better study of the arrangement of the internal structure of the cells, as a too deep colorization does not sufficiently differentiate the chromophilic granules and network. The outer surface of the cortex is here seen to be smooth and regular. Immediately beneath is the first or superficial layer, pale in color, .25 mm. in thickness, and characterized by a paucity of cells irregularly arranged and appearing upon a colorless background, which we know from sections treated by the Weigert method to be made up of large numbers of densely packed nerve fibers and processes. In the plate are seen for the most part neuroglia cells, but under a high power here and there nerve cells are found, rounded, spindle-shaped, or polygonal in form, with a nucleus filling up almost the entire cell-body, and with no visible dendritic processes, or at most but one or two extending vertically or at right angles to the surface of the cortex. The cell-body contains but a small mass of chromophilic substance, usually forming a narrow band about the nucleus with thickenings where the dendritic processes are given off. The cells with no visible processes of course may have such extending in a direction outside of the plane of the section. Fig. C shows one of these cells containing no visible processes, with a rounded cell-body, a large rounded nucleus almost filling up the same and surrounded by a uniformly thin band of chromatic substance. Fig. D shows another cell from

this same region with a single process vertical to the surface of the cortex. Here again the large rounded nucleus almost fills up the cell-body and is surrounded by a thin band of chromatic substance thickened at the point where the dendritic process is given off, and extending into the same for a short distance. Fig. E shows a similar cell, but with two processes extending in a nearly horizontal direction. The thin chromatic band is here thickened at both extremities. The greatest diameter of the cell in Fig. D is vertical, while in Fig. E it is horizontal, in Fig. C of course being practically uniform. Occasionally cells are seen with one process vertical and the other horizontal, as in Fig. F, this being polygonal in shape. The nuclei in all of these cells are well defined, large, and contain a small amount of chromatin in the form of a poorly differentiated network, and at some point containing a distinct nucleolus. According to Nissl's classification of nerve cells these would be known as karyochrome nerve cells. The neuroglia cells are smaller, rounded or oval in shape, contain a nucleus, and are much more numerous in this layer than the comparatively scarce nerve cells. The second or pyramidal cell layer, to simplify the layering of the cortex to the greatest extent, as previously described, is made to include both the large and small pyramidal cells. This measures in the plate (Plate II, Fig. 3) 1.40 mm. in depth. This layer thus includes the large and small pyramidal cell layers and the ganglionic cell layer of Hammarberg, and the large and small pyramidal cell layers of Golgi and of Cajal. (See Table I.) Hammarberg (1895) in his description of this region states that the layer of small pyramidal cells does not form a distinct layer, but gradually passes over into that of the third layer of large pyramidal cells, so that the two in his division make a layer .80 mm. in depth. He further states that the under border of the third layer is difficult to determine, so that there is no distinct fourth layer. This fourth layer is then described as containing small pyramidal cells less thickly distributed than in the layer above, intermingled with which are some smaller irregular cells, giving the appearance of a region poor in cells; and he notes further that in many places the difference is so slight that it does not make a separate layer. His fifth layer is described as a .70 mm. thick ganglion cell layer, made up of somewhat larger and more closely packed pyramidal cells. These layers,—second, third, fourth and fifth layers of Hammarberg, are all included in this second layer of the writer, and in this section measures about 1.40 mm. in thickness. It is made up of pyramidal cells varying in size in all parts of the layer, so that in any region, excepting a band below the middle portion to be described later, pyramidal cells of different size may be seen in close proximity.

As a rule, however, the larger pyramidal cells are found mostly in the deeper portion of the layer. But here as elsewhere are to be seen the small pyramidal cells interspersed. There are also to be seen some rounded and irregular cells throughout this layer. Just below the middle of this second layer is seen a belt some .30 mm. in width, in which small pyramidal cells decidedly predominate and containing but few larger and medium sized pyramidal cells. This region is comparable to the fourth layer of Hammarberg, but as it contains no elements differing from the region above and below, but simply varies in the relative number and size of the same, it does not seem to the writer worthy the designation of a separate layer with the resulting increase in the number of layers. Both above and below this belt large and small pyramidal cells are intermingled; the large pyramidal cells increasing in proportion from above downward. Fig G (G of Plate II, Fig. 3) shows the details of structure of one of these small pyramidal cells under a high magnification ($\times 1,300$. No. 3 ocular, 1-12 in. oil immersion objective, Leitz). By focussing in different planes the large, broad, apical, dendritic process is seen extending vertically towards the surface of the cortex. It contains chromophilic granules arranged with the long axis parallel to the axis of the cell. The cell-body is in the form of a pyramid with rounded sides. The large nucleus is irregularly oval with the long axis parallel with the long axis of the cell. The nucleolus is centrally located and surrounded by the slightly stained nuclear contents. From the lower portion of the cell-body are given off three dendritic processes, one of which subsequently branches into two processes, at which point a mass of chromatin is seen, one of the so-called "wedges of division" (Versweigungs-Kegeln) of Nissl. These dendritic processes contain no chromophilic granules, but at the point where they are given off from the cell-body are found aggregations of chromophilic granules which also surround the nucleus on either side, their long diameter extending in general parallel with the long axis of the cell. Plate II, Fig. 4, is a photomicrograph of this same cell as stated, indicated by the letter G in Plate II, Fig. 3, and shown in Fig. G of the text as already described. In order to secure a focus by which the cell processes could be seen even faintly the nucleus and nucleolus are almost entirely out of the plane of focus, the latter showing faintly however. Immediately about the nucleolus there is a slightly pale area, a portion of the nucleus. The contents of this cell-body itself present for the most part the appearance of a very dark, almost homogenous, intensely stained chromatic substance, and gives only a partial idea of the real structure and arrangement of the granules as seen in Fig. G. The contour of the nucleus

cannot be distinctly made out, nor the relative position of the nucleolus in the same. The individual chromophilic granules cannot be distinguished excepting at the base of the right lateral dendritic process. The apical dendritic process appears as a mere shadow, owing to its being out of the plane of focus. The basal dendritic process show almost as well as in Fig. G, excepting the branching of the one at the left which is but faintly indicated. The small mass of chromatin at this point—the so-called “wedge of division,” of Nissl—is also but faintly indicated. The



Plate II, Fig. 4.

general contour of the cell, points from which the dendritic processes are given off, and position of the nucleolus are accurately represented. Had a plane been selected showing the nucleus accurately some of the processes would have been entirely out of focus. Other cells adjacent to the one just described are seen to be less deeply stained and the chromatin is seen in the form of larger and smaller indistinctly rounded or elongated granules. It will thus be seen that there are limitations, and serious limitations too, in the use of photomicrographs, especially under high powers, in such a study as this. It would require several photomicrographs at various planes to bring out

all the details shown in the single Fig. G, which of course is a composite taken by focussing in all planes under a magnification of 1,300 diameters, and showing certain, but not all, details of the different planes. Of course outlines are not so distinct in the real cell as must necessarily be made to appear in a drawing, and this latter might be compared to a dissection in which certain parts are brought out prominently at the expense of the normal appearance of the whole in life. No one would hesitate to say, however, that Fig. G gave a much better idea though not an exact representation of the cell, than Plate II, Fig. 4, and that drawings are essential accompaniments of such plates in the full elucidation of a subject of this kind. These cells are known as stichochrome nerve cells of the somatochrome class. Fig. H shows one of the large pyramidal cells (the largest to be seen in the plate) under the same magnification as in Fig. G ($\times 1,300$. No. 3 ocular, 1-12 in. oil immersion objective, Leitz). The internal structure of the large pyramidal cells is found to be made up of distinctly stained chromophilic substance in larger and smaller irregular granules, some so fine and closely arranged as to completely fill up the cell-body, thus presenting a uniform field of closely packed minute granules without any special relation to one another. In other cells the granules are coarser and present a somewhat parallel arrangement about the nucleolus and extending up into the main apical process for some distance and also into the basal lateral processes in the same general parallel manner. In other pyramidal cells there is a fine chromophilic network with larger and smaller aggregations of chromatin as nodes or enlargements of the network. In this figure the cell-body is pyramidal in shape with rounded contour merging above into the large apical dendritic process, which is finally lost to view as it enters another plane. Below at the base, both to the right and left, are given off several dendritic processes, some of which are seen to branch a short distance from the cell-body. The large, slightly oval nucleus is centrally situated somewhat nearer the base of the cell than the apex, and contains a large, eccentrically placed nucleolus, surrounded by the pale, slightly strained, for the most part homogenous, nuclear protoplasm. Within the cell-body about the nucleus and extending into the apical process, and to a less extent into the basal dendritic processes, are chromophilic granules of rounded or elongated shape, and having a tendency to be arranged in groups in places. Especially at the point where a dendritic process is given off is frequently to be found a mass of these granules. Often a wedge or cap of chromophilic substance is seen at the point where a dendritic process is to be given off, as at *a*, or where a process divides into two branches,

as at *b*, the latter being one of the so-called "wedges of division" of Nissl. The granules vary in size and shape and are arranged in general in a direction parallel to the long axis of the cell-body and of the long axis of the dendritic process when extending into the same. About the nucleus they are often arranged parallel to its wall. Five dendritic processes are given off from the base of this cell, three of which are seen to divide into two

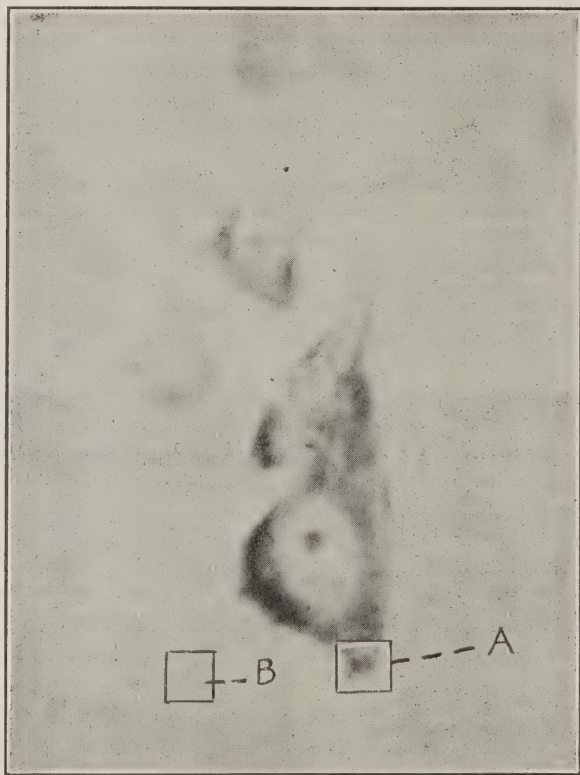


Plate II, Fig. 5.

branches each; at *b*, showing one of the so-called "wedges of division" of Nissl, referred to above. Plate II, Fig. 5 is a photomicrograph of this same cell indicated by the figure H in Plate II, Fig. 3, and already described and shown in Fig. H; here under a magnification of 1,400 diameters. The body of another somewhat smaller pyramidal cell is seen just behind the apical dendritic process of this cell and part of it almost in

the same place, so that it is difficult to differentiate the two. It will be seen that the nucleus and nucleolus are in good focus, but that the basal dendritic processes, excepting at their origin from the cell-body, can scarcely be distinguished. The shape of the nucleus, size and position of the nucleolus, and arrangement of the chromatin within the same is as well shown as in the figure. Also the shape and contour of the cell-body. The dendritic processes, however, as mentioned above, are out of the plane of focus, so that only a very hazy, indefinite indication of the beginning of three of them can be made out. The large one at the left can scarcely be determined at all. The more central one at the left is faintly outlined, and at the point of bifurcation the so-called "wedge of division" of Nissl (B) is seen, but the division itself cannot be determined. Of the three processes given off from the base at the right the beginning of the one most centrally located can be very faintly seen and a faint outline of the other two, appearing as one in the photomicrograph is seen. The apical dendritic process is so merged with the body of the cell just below it that it is difficult to determine its outline. The cell protoplasm is more differentiated however than in the preceding plate (Plate II, Fig. 4). Here chromophilic bodies are seen at the base on the right where the dendritic processes are given off, indicated by A both here and in Fig. H. Chromophilic granules are also seen fairly well indicated to the right and above the nucleus, in larger and smaller bodies without sharp outlines, which is the condition actually found in the cell. They are not sharply and definitely outlined as shown in Fig. H, which in that respect is not accurate, but merely diagrammatic. At the left and below the nucleus in the basal portion of the cell-body these chromophilic bodies are so closely aggregated as to give almost a homogenous dark appearance to that portion of the cell, and it is only by carefully focussing in various planes that the chromophilic bodies are made out. Here again the photomicrograph without the accompanying drawing would give one only a partial knowledge of the structure of this cell. Cells of this type are to be classified as large stichochrome nerve cells of the somatochrome class. Interspersed among these larger and smaller pyramidal cells in this layer are some small irregular and rounded cells consisting of a large nucleus containing a nucleolus, and surrounded by a cell-body containing in the smaller cells a very narrow rim of finely granular chromophilic substance, in some places so narrow as to be scarcely discernible. In other cells the cell-body is much more developed surrounding the nucleus and giving off several protoplasmic processes which go out in various directions. Neuroglia cells similar to those of the first layer are

found interspersed among the nerve cells in all parts of the section. Small portions of capillary blood vessels are shown as at 1 in the plate (Plate II, Fig. 3, 1) where the vessel is seen to divide into two branches below, and also at 2 (Plate II, Fig. 3, 2), showing a single small capillary extending only for a short distance in this plane. The walls are seen to be exceedingly thin, made up of a single layer of nucleated cells, and containing red blood corpuscles. The walls of these blood vessels stand in marked contrast to the thickened and tortuous walled vessels found in the cases of dementia paralytica later to be described. Below this is the third, or spindle cell layer about 1.20 mm. in thickness in this section. The spindle cells in this layer contain a large nucleus, with a distinct nucleolus, but no nuclear net work could be made out. The nuclei in many of them present the appearance of being too large for the cell-body, so that often, as in Fig. I, the cell-body seems to be bulged out on one side to accommodate the large nucleus, giving an eccentric form, and resulting in a spindle with one side very prominent and the opposite side quite flattened. The chromatic substance is arranged in no definite network and contains no distinct chromophilic bodies, but consists of minute particles closely aggregated about the nucleus, especially at each pole and found in a lesser amount and in variable quantities in the remainder of the cell-body. In Fig. J the nucleus is situated eccentrically at one end of the cell-body, so that most of the other cell contents are at the opposite end with a narrow band of closely aggregated chromophilic substance surrounding the nucleus at both ends. The nucleus in this cell is more elongated and oval in shape, with distinct nucleolus and otherwise pale, slightly stained contents. Other cells as Fig. K are more or less irregular in shape, in this case having the form of an irregular inverted pyramid containing a large rounded nucleus near the base which is surrounded by a narrow band of dense chromatic substance. About this is an irregular chromatic network containing nodal thickenings at some points. The nucleus contains a distinct nucleolus, but otherwise pale and but slightly stained contents. A large dendritic process is seen given off below and two dendritic process go off from the base and to one side. Fig. L shows another irregular shaped cell with a dendritic process given off from one side in addition to those from each end. The large oval nucleus with distinct nucleolus and otherwise pale contents is centrally placed. Considerable chromatic substance is seen in the larger cell-body with distinct granules, forming an indistinct network at the point where the lateral dendritic process is given off, on the opposite side and also in the cell-body and at the base of each of the vertical pro-

cesses. Plate III, Fig. 6, is a photomicrograph, at a magnification of 1,400 diameters, of a group of the spindle and irregular cells indicated by the letter I in Plate II, Fig. 3.

As will be noted, almost all the forms shown in the above described figures are to be seen here. The cell lettered I, is similar in shape and structure to that of Fig. I, including the bulging out of the nucleus to one side, and showing an aggregation of chromatic substance in the form of a nuclear cap at both poles of the nucleus. An irregular network of chromatin

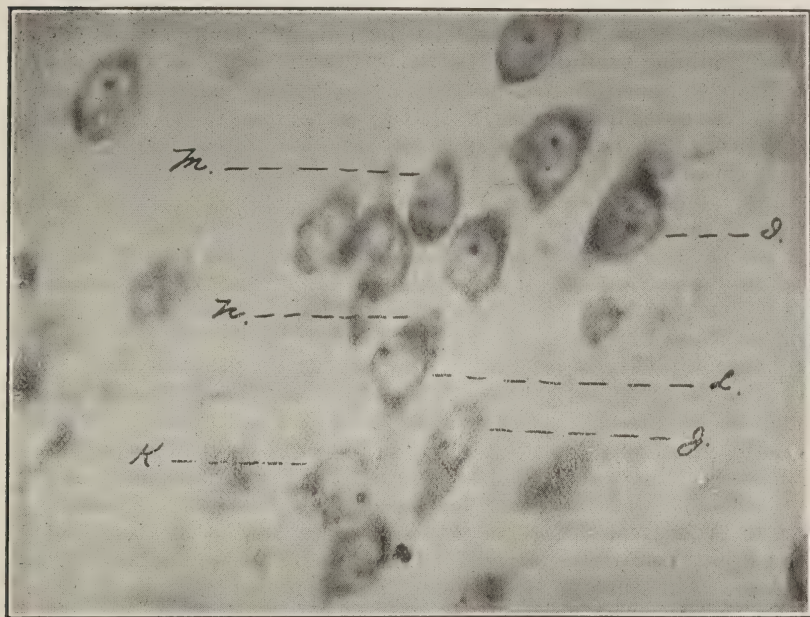


Plate III, Fig. 6.

is seen within the nucleus surrounding the nucleolus. But little chromatic substance, in addition to the two nuclear caps above described, is to be seen in the small cell-body. Again the cell indicated by the letter J is very similar to that seen in Fig. J, the nucleus being at one end of the cell-body near the base of that polar dendrite. The nucleolus is situated at the lower portion and to the left in the nucleus and with but little faintly stained chromatic substance surrounding it. About the nucleus in the cell-body and extending into both dendritic processes is to be seen considerable chromatic substance arranged in an indefinite network. The cell lettered K is irregularly pyramidal

in shape, approximating to that seen in Fig. K. The nucleus is very large and almost entirely fills up the cell-body. The nucleolus has the appearance of being without the nucleus, but is within the same adjacent to the wall at this point. A small amount of chromatin is seen in the lower part of the nucleus, otherwise it is for the most part pale. A process is seen given off above as the apical dendrite at the base of which are some few chromophilic granules.

Two basal dendritic processes are given off, one to the right and projecting almost vertically downward, and the other to the left and almost horizontal in direction. At the base of these processes, especially the one to the left, are seen well marked chromophilic granules. Otherwise the cell-body is practically entirely taken up by the nucleus. The cell indicated by the letter L is somewhat similar to that of Fig. L in regard to having two polar dendritic processes, one of which coming off laterally here on the left side and only faintly indicated, being in a lower plane. The nucleus here also is seen to occupy the greater portion of the cell-body, the nucleolus being crowded over almost to the edge of the same. The nucleus contains only a slight amount of chromatic substance in its upper portion. Nuclear caps are seen above and below the nucleus in the cell-body, the one above being the larger, and surrounded by some small chromophilic bodies extending up into the dendritic process. A very slight amount of very palely colored chromatic substance is seen below the nuclear cap in the lower dendritic process. The lateral dendrite when seen in focus is found to contain a slight amount of chromatic substance more abundant at the base. The cell lettered M is regularly spindle in shape with two polar dendritic processes, the lower one not in the plane of focus, and the upper one only partially so, and there are no lateral processes. The nucleus is not in distinct focus, so that its outline is indistinct. The nucleolus is seen at the lower part of the nucleus, and to the left side surrounded by a rather pale indefinite chromatic network. Below this is seen some dense chromatic substance in the form of a nuclear cap. The nucleus really occupies almost the entire body of this cell extending from the dark mass below which has the form of an irregular nuclear cap, to the base of the upper dendritic process where there is a smaller nuclear cap of chromatic substance. A small amount of protoplasm above and below the nucleus in the cell-body extends for a short distance into the dendritic process from these nuclear caps. The other cells seen in this plate are similar in appearance and structure to those already described.

(Plate illustrations referred to and not found will appear in the November issue.)

(*To be continued.*)

Periscope.

NOUVELLE ICONOGRAPHIE DE LA SALPETRIERE

(Vol. 16, 1903, No. 1, January-February.)

1. Chronic Hypertrophic Acrocyanosis. M. PÉHU.
2. A Case of Progressive Facial Hemiatrophy. CALMETTE and PAGÈS.
3. Acquired and Progressive Chronic Trophoedema. J. SICARD and LAIGNEL-LAVASTINE.
4. A Case of Macroactylia. F. LEJRAS.
5. Macroactylia. CAYLA.
6. In Regard to the So-Called Sense of Position. CLAPAREDE.
7. Les Urologues. HENRY MEIGE.
8. A Note Upon Three Designs of Jordæns. JEAN HEITZ.

1. *Chronic Hypertrophic Acrocyanosis*.—The pathology of the extremities, acropathology, is yet a limited field in medicine. The types at present known are Raynaud's Disease, L'Osteoarthropathie hypertrophiant pneumonique of P. Marie, and Acroparesthesia of Schultz. The characteristics of these processes are the localization and the sensory vasomotor or trophic type of the symptoms. It is not always possible to differentiate sharply each variety from the others on account of their complexity. In addition to Raynaud's disease, there are three main types in which asphyxia is constant and chronic; they are acrocyanosis with sensory symptoms, with atrophy, and with increase in the volume of the soft parts. The author of this paper, after giving a literature resumé of cases previously described, gives one of his own which evidently belongs to the third class. Case.—Young man, twenty-five years of age, neuropathic history, alcoholic, tuberculous, presented during a period of ten years' intermittent attacks of cyanosis of hands and feet. The asphyxia became in time permanent, and was accompanied by pain so severe that he had to give up his daily work. In addition, the hands increased in volume without any corresponding lesion of the bone (radiograph). There were never any sensory disturbances and no attacks of local syncope. The following were considered in the differential diagnosis: L'osteothrop hypertrophiant pneumonique of P. Marie, acromegaly, acroparesthesia, erythromalalgia, and Raynaud's disease.

2. *Facial Hemiatrophy*.—A case of progressive facial hemiatrophy in a young girl, fifteen years old. There are two theories to account for this disease: First, primary atrophy of the subcutaneous tissue; second, a lesion in the facial nerve, sympathetic, or the trigeminal, the so-called nervous theory. Brissaud's theory seems more reasonable. "In the presence of a hemilateral affection, involving the seventh, fifth and twelfth nerves, it is natural to think of a lesion in the cerebrospinal axis. "This paper is illustrated by some remarkable photographs.

3. *Chronic Trophoedema*.—A classification of edemata based upon the evolution of the symptoms, and not upon the etiology or pathogenesis, seems with our present knowledge the most simple and effective. Henry Meige, in a series of studies, has described a type to which he has given the name of chronic trophoedema. The following divisions and subdivisions are at present recognized: 1. Acute Edemata: (a) infectious, (b) neuropathic; 2. Hysterical Edemata; 3. Chronic Edemata: (a) depending upon various chronic diseases of the heart, lungs, nervous system, etc.; (b) chronic edema of elephantiasis; (c) tropho-edema (Meige). This paper is based upon a case of chronic acquired tropho-edema. Girl,

twenty-seven years of age, neuropathic heredity; as a result of a trauma of the left foot a painful edema appeared, localized at the site and a little above the contused region. This edema was at first transient, but gradually became permanent, and progressively involved the leg, thigh, buttocks, of the same side. Process was a slow one, taking in all two years.

4. *Macroductyly*.—A case of local gigantism in a child thirteen years old. The median finger of the left hand was enormously enlarged. There was no hereditary history. This finger measured 15 cc. in length, 12 cc. in circumference in the middle of the first phalanx; finger was amputated. The article is illustrated by radiographs and photographs.

5. *Macroductyly*.—A further case of macroductyilia in a boy seventeen years old.

6. *Sense of Position*.—A discussion of Bonnier's paper on the sense of position, which appeared in No. 2, 1902, of this Journal. (Not adapted to abstract.)

7, 8. Historical and literary notes on medicine in art.

SCHWAB (St. Louis).

(Vol. 16, 1903, No. 2, March-April.)

1. Syringomyelia; Arthropathy of the Shoulder (Muscular Atrophy and Thermoanalgesia of a Transverse Type). E. BRISSAUD and BRUANDET.
2. An Old Lesion of the Red Nucleus. Secondary Degeneration. PIERRE MARIE and GEORGES GUILLAIN.
3. A Case of Late Hereditary Syphilis of a Cerebrospinal Form. RICHON.
4. A Case of Symmetrical Sclerosis of the Occipital Lobe. L. MARCHAND.
5. A Case of Progressive Myopathic Atrophy, with Sensory Symptoms. M. LANNOIS and A. PAROT.
6. Enormous Deformity of the Right Tibia (Forreau de Sabre) in a Tabetic. J. SABRAZES.
7. A Case of Phocomelia and Hemimelia. P. HALBRÖN.
8. Some Examples of Edemata in Art. HENRI MEIGE.

1. *Syringomyelia*.—Man, forty-nine years, with tuberculosis of the lungs and a scapulo-humeral articulation very much deformed and augmented in volume. The diagnosis of syringomyelia was based upon the arthropathy, muscular progressive atrophy of the left upper extremity, and a dissociation of sensibility localized in both arms. The man died of tuberculosis. Microscopical examination of the cord showed typical appearances of syringomyelia. The shoulder joint showed trophic changes of a very interesting nature. The articular capsule had disappeared together with the ligaments. The long head of the biceps was absorbed; its insertion in the glenoid cavity could not be recognized. Villous vegetations filled the joint. In the muscles of the subscapular, supra, and infraspinous regions ossiform nodules could be demonstrated.

2. *Lesion of Red Nucleus*.—An old lesion found in the red nucleus and causing secondary degeneration of various nerve tracts gave the subject matter of this paper. On account of the rarity and limitation of the lesion, a study of sections above and below it seemed to promise interesting results. The subject of this study had infantile cerebral palsy, from which a left hemiplegia remained. At the age of twenty-seven years he became infected with syphilis, death occurred at forty-nine, with progressive dementia. No cortical or meningeal lesions could be demonstrated. On the left side, exactly in the red nucleus, a lesion was found. It was either an old tubercle, which had passed into the sclerotic state, or an old limited focus of softening. Posteriorly and anteriorly to the red nucleus the structures are normal, showing the sharp limitation of the lesion. It could be compared in fact to a veritable experimental destruction of the red nucleus.

Cross sections above and below the lesion showed very interesting fiber degenerations, the most important of which are degeneration of the cerebellar peduncle, the central tract of the calotte, and the posterior longitudinal bundle. Very excellent plates illustrate the article.

3. *Tardy Syphilis*.—This is a very careful histological study of a case of tardy congenital syphilis in which the lesions were localized chiefly in the cerebrospinal system. The etiological diagnosis could only be made from a study of the lesions. Boy, age eleven years, without visible lesions or stigmata of inherited syphilis. Fistulous lesion of the parietal bone accompanied by Jacksonian epilepsy. Unsuccessful surgical intervention. Persistence of the epileptiform convulsions, accompanied by hemiplegia, with contracture. Progressive decline in intellect, deafness, blindness. Cachexia and terminal tuberculous infection. Autopsy: Fibrous meningo-encephalitis, basal gummatous meningitis, cavity formation in the cerebellum, infiltration of spinal meninges. Examination of the skull showed evidences of an extension of the fibrous encephalitis into the bony substance. The evolution of this condition is thus summarized: The brain already structurally deformed, with probably a certain degree of hydrocephalus, a condition not rare in congenital syphilis, became the seat of a gummatous neoplasm. A gumma arising from the periosteum, or osseous tissue, perforated the inner table and attacked subjacent tissue, producing adhesions. The meningeal encephalitis developed slowly at first and led to a sclerosis of the hemispheres, causing the symptoms of cortical irritation, that is the epilepsy, and later the hemiplegia. The lesions then became more diffuse, extended to the cord, the base of the brain and to the white matter of the cerebellum. The specific nature of these lesions is very evident on account of the evolution of the process and their anatomical character, as well as by the existence of a veritable syphilitic stigma in the cerebral malformation.

4. *Sclerosis of Occipital Lobe*.—This case is interesting not only because it confirms the previous work on visual localization, but because it determines the group of symptoms which follow a symmetrical sclerosis of the occipital lobe. The slow course of the disease, and the definite lesion leading to a complete destruction of the occipital lobe add particularly to the interest of the case. Girl, nineteen years old; epilepsy; almost complete blindness; central vision for light alone remains, but the patient cannot recognize objects. The eyes are normal. Patient died in status epilepticus. Autopsy: The external and internal surfaces of both occipital lobes are covered by a membrane penetrating into the cerebral substance. The pia mater is adherent. The convolutions are much atrophied. The internal surface presents the same appearance. Microscopical examination shows sclerosis with numerous foci of softening and a great increase in neuroglia. The preservation of a visual field to light around the point of fixation in this case is of great interest, and seems to uphold the theory of V. Monakow, to the effect that the fibers corresponding to the macula are distributed in the whole extent of the cortical field of vision.

5. *Myopathic Atrophy*.—The distinction between muscular atrophy of myopathic and nervous origin is gradually being given up, and an intermediate variety, which includes many irregular forms, is being substituted. Various transitional types belong here. Such a one forms the subject of this paper. Clinical résumé: Man, thirty-eight years old; no family disease; neuropathic heredity; traumatism at seven years; first appearance of symptoms at ten years by lancinating pain and paresis of the muscles. Slow progressive development with a persistence of the pain and pseudohypertrophy of the lower extremities; muscular atrophy in all four extremities, symmetrical in respect to nerve root distribution and almost total in respect to segmental distribution; face not involved. Pseudo-hyper-

trophy of lower extremities, tendon contractures, abolition of tendon reflexes; almost complete disappearance of electric excitability, subjective and objective sensory disturbances.

6. *Tabetic Arthropathy*.—Cases of tabes with evidence of syphilis other than the metasyphilitic nervous lesions, are always of great interest. A number of cases belonging to this variety are increasing rapidly. The case here described is that of a man, forty years old, symptoms of tabes appearing at thirty-eight, as follows: Gastric crises, ocular symptoms, diplopia, Argyll-Robertson pupil, abolition of knee reflex. No history of acquired syphilis. Evidence of hereditary syphilis is present in the characteristic dental malformation and in a sword-like deformity of the right tibia (En Fourreau de Sabre). The character of the tabes in this case is very similar to that which is found in infantile tabes depending upon hereditary syphilis.

7. *Phocomelus*.—Description and photograph of a case of Phocomelia and Hemimelia.

8. An Art Study.

SCHWAB.

JOURNAL DE NEUROLOGIE

(1903, VIII, No. 5.)

1. The Differential Diagnosis of Epilepsy. KOWALEVSKY.

2. Incipient Tabes. DE BUCK.

1. *Epilepsy*.—In a very complete review, the author compares the symptoms of epilepsy with those of apoplexy, hysteria, eclampsia and convulsions from other sources, the parietic seizure and vertigoes. Sketching the characteristics of epileptic psychical disturbances, he gives the chief points distinguishing them from acute mental symptoms due to other causes. He then considers the characteristics from a diagnostic and prognostic point of view, of epilepsies of different kinds with the special indications for their treatment. Of particular interest are his remarks on epilepsy of syphilitic origin.

2. *Incipient Tabes*.—The author exhibited at a meeting of the Belgian Neurological Society, the case of a man of thirty-one years, without alcoholic or syphilitic history, but acknowledging *abusus Veneri*, who presented the following symptoms: Fatigue in walking, slight dragging of the feet, with dysesthetic sensations in the lower limbs, slight diminution of the pain sense in the area of distribution of the second sacral segment, slight ataxia and Romberg, exaggeration of the patellar and arm reflexes, loss of the plantar, Achilles tendon, cremasteric and pharyngeal reflexes. His pupils reacted to light and accommodation. No change in the electric reactions, sphincters normal, constant genital irritation with tendency to priapism. The diagnosis of incipient tabes he bases upon the sensory troubles, and especially upon the loss of the Achilles tendon reflex, and quotes a number of authorities in support of this view of the case.

ALLEN (Trenton).

(Vol. 8, 1903, No. 6, March 20.)

1. Tuberculous Polyneuritis with Exaggeration of the Tendon Reflex in the Rabbit. D. DE BUCK.

2. Infantile Cerebral Palsy. BASTIN.

3. Tic. DE BUCK.

1. *Tuberculous Polyneuritis*. It is well known that in multiple neuritis, the tendon reflexes are occasionally found to be exaggerated. This has been accounted for in three ways: (1) As due to increased irritability of the centripetal fibers (the nerve not being totally degenerated); (2) as due to increased irritability of the muscular fibers, and (3) as due to the

action of the poison of the disease upon the nerve cells in the cord, increasing their irritability. The author, after reviewing briefly these theories, as put forward by different writers, describes his findings in the case of a rabbit affected with neuritis in the posterior extremities, in the course of a general tuberculosis, in which the tendon reflexes were exaggerated. The muscles of the affected limbs were practically normal, the nerves showed marked parenchymatous neuritis, and there was diffuse degeneration of the columns of the cord. The cells of the anterior horns, and of the spinal ganglia, on the contrary, were normal. These findings seem to dispose of the muscular theory, and to give little support to that of cell irritation, but point rather to increased irritability of the nerve fibers as a cause of the symptom. However, the author thinks that the question as to the cutting off of inhibition by the severing of the paths from the superior to the inferior nerve centers, is to be considered, on account of the degeneration found in the pyramidal tracts.

2. *Infantile Cerebral Palsy*.—A case, history and demonstration, bringing out nothing specially new.

3. *Note on Case of Tic*.—The demonstration of a case of involuntary movements of the facial neck and shoulder muscles. The author diagnoses the case as one of tic, and in connection with it discusses the pathogeny of this disease and of the allied conditions of myoclonus, chorea and athetosis. He seeks to explain the difference between true tic and the other conditions in question in the existence of two sets of cortico-spinal fibers, the pyramidal, or direct, and the extrapyramidal, or cortico-mesencephalo-spinal. In true tic (or mental tic, as he calls it) the impulse passes from the psychomotor region of the cortex by the direct way. In other involuntary movements, on the contrary, it passes down by the extra-pyramidal tracts, or depends upon a reflex irritation radiating from the spinal cord. The author some years ago formulated the theory that the localization of the irritation giving rise to "clonic involuntary hyperkinesias" should be found in the basal ganglia or in the extrapyramidal motor fibers descending from them. Proof of the correctness of this has, in the meantime, been accumulating, he thinks, and he refers in this connection to the papers of Redlich, Probst, Rothman, Sorgo, and others, who have studied the results of lesions in this region.

(Vol. 8, 1903, No. 7, April 5.)

1. Circular Insanity and the Circular Forms of Psychoses. S. SOUK.
HANOFF and P. GANOUCHE (also see *Arch. de Neurologie*, May, 1903).

2. Medical Graphology. MLE. D. PEPPEE.

1. *Circular Insanity*.—The author gives the results of a study of 86 cases of circular insanity from the Moscow Psychiatric Clinic, and draw the following conclusions. This form of insanity is nearly three times more frequent in women than in men; it constitutes 1.94 per cent of all the cases (4434) received. It is twice as frequent as mania, and only one-third as common as melancholia. In its etiology, heredity is quite as important as in mania, in which it was found to play a rôle in 84 per cent of all cases. Two-thirds of all cases of circular insanity begin between the ages of sixteen and twenty-five years. In the majority of cases the disease is ushered in by depression. They then take up the question of the occurrence of psychoses in circular forms, and narrate some interesting cases, in some of which the disease, though showing alternating phases, was evidently due to organic changes in the brain.

2. *Medical Graphology*.—A discussion, from a diagnostic point of view, of the characteristics of handwriting. The author, a handwriting expert, makes certain suggestions as to conclusions as to character and state of

mind of an individual which may be drawn from a study of his or her handwriting.

(Vol. 8, 1903, No. 8, April 20.)

1. The Patellar Reflex in Neuroses. L. SCHNYDER.

2. Dolorific Assymetry. J. IOTYKO and M. STEPHANOWSKA.

1. *Patellar Reflex*.—The author calls attention to the extreme difficulty in securing relaxation of the limb when examining the condition of the patellar reflex in nervous subjects, the patients usually seeming unable to keep from contracting either the extensor or the flexor muscle of the leg. He regards this as constituting a symptom of some value as indicating nervous irritability, and compares its method of production with that of the muscular rigidity found in myotonia congenita, in catalepsy, and the negativism of certain mental diseases, and in hysterical contraction. To illustrate his views he gives short clinical histories of ten patients suffering from neurasthenic, hysterical and mild mental symptoms, in whom this phenomenon was present.

2. *Dolorific Assymetry*.—It has long been known that there is a difference in general sensibility upon the two sides of the body, greater upon the right in right-handed persons, and upon the left in left-handed ones. Van Biervliet has shown that the proportion is usually as 10 to 9. Starting out from the above facts, and from the researches of Goldscheider and Von Frey, which seem to show proof that there are special nerve fibers, respectively for pressure, for pain, and for temperature sense, the authors carried on a series of experiments with a view of ascertaining if there is any difference in the acuteness of the pain sense upon the two sides. In their measurements they made use of the algometer of Chéron. Their experiments were performed upon 52 subjects, mainly university students, fourteen of this number being left-handed. The points chosen for pricking by the instrument were exactly symmetrical areas on the anterior surfaces of each forearm, just above the wrist. The following are the results obtained: Two of the subjects were practically insensible to pain. Of the remaining fifty, three persons showed greater sensitiveness upon the right side, but in two of these even, the result was doubtful. All of the remaining forty-seven showed greater sensibility to pain upon the left side, both right-handed and left-handed persons being included in the number. Taking the average of the elgesimeter readings for all of these, the sensibility of the two sides was found to be as 10 to 9 in favor of the left, conforming to the figures of Van Biervliet. Experimenting upon other regions (the finger tips, back of the hand, temples), the authors still found sharper pain sense in these cases always on the left. They make no attempt to explain the cause of the difference.

C. L. ALLEN, Trenton.

PSYCHIATRISCHE, NEUROLOGISCHE, WOCHENSCHRIFT

(Vol. 4, 1903, No. 48.)

The Treatment of Epilepsy According to the Toulouse-Richet Method.—Halmi and Bagarus. Fifteen cases of epilepsy which had been under observation in the hospital for some time were selected for the investigation. For ten months these patients were treated with 3-5 gms. of bromide daily, then followed two months without any medicinal treatment, at the end of which time the Toulouse-Richet method of treatment was applied for one month. During the first half of this period 3 gms. of bromide were given, when the dosage was reduced to 1.5 gms.; diet consisting of 2 litres of milk, 2 eggs, and for the men 750 gms., for the women 500 gms. of salted bread. Following this period of treatment they returned to the simple bromide treatment. In view of the great variation in the number of attacks monthly during treatment with simple bromides—in one case

19-31, in another 3-104 attacks, it would seem manifestly unfair to accredit a cure unless there was improvement in a large majority of the cases. Furthermore, in the nine months without change of treatment, nine cases showed spontaneous improvement. During the two months when the bromides were withdrawn the attacks increased in seven and diminished in three cases, and two patients died, but the total number of attacks fell to 273, or nearly one half of the total number in the two months when the bromides were withdrawn. Still, during six of the ten months in which the simple bromides were given, the total number of attacks was less than during the test month. Furthermore, a close study of the individual cases which seemed most improved reveals the fact that just as great improvement had previously occurred during the treatment with the simple bromides. In the final period of seven months the total number of attacks in the thirteen remaining cases rose to 324, but later fell to 219, in spite of the fact that the bromides were entirely withdrawn in five cases. Besides this doubtful effect in the number of the convulsions, this method of treatment had an appreciable sedative influence upon all psychical processes. The patients were less restless and exhibited a retardation which, in some cases, almost reached genuine stupor. In two cases it produced a fatal intoxication. In conclusion, the Toulouse-Richet method neither cures nor benefits, and greatly enhances the danger of bromism.

(Vol. 5, 1903, No. 1.)

The author (Waltenberg), after citing the theoretical and practical reasons for the abolishment of the "isolation cell" in the treatment of maniacal patients, presents the results accomplished during the past six years (1896-1902) at Lübeck, by the substitution of the constant watch, and the prolonged bath. He observes that during this period a greater degree of quiet prevailed, and there was a proportionate less demand for narcotics. The plotted curves accompanying his paper show the gradual fall of the percentage of women reported restless at night (11.3 per cent to 3.2 per cent), and a similar fall in the use of narcotics (3.6 per cent to 1.2 per cent).

(Vol. 5, 1902, No. 2.)

The Prognosis and Therapy of Intemperance. SAUERMANN-BONN.

The author calls attention to the increasing prominence of institutions for the treatment of inebriates, the reports of which record from 15-80 of recoveries, to the prominent rôle played by the "after treatment" in the third conference of the German inebriate institutions, and cites the method instituted at the Swiss Institution Ellikon by the establishment of a society of discharged patients for the purpose of mutual aid in maintaining abstinence. Their results are 70 per cent of recoveries from 1896-1900. His statistics relative to the prognosis are based upon 246 replies from members of the Red Cross Abstinence Society. He concludes (1) that recovery from alcoholic intemperance is synonymous with prolonged abstinence; (2) the frequency of defective heredity and a psychopathic constitution has led to the belief that a large number of alcoholics are incurable. The real basis for this belief lies in a lessened resistance to the action of alcohol; the drink custom and other accompanying influences only give opportunity. The patient cannot be cured of hereditary intolerance, he can only be freed from alcohol. Recovery from acquired intolerance is possible. (3) The necessity of prolonged abstinence and the great tendency to relapse demands prolonged attention after dismissal from treatment. (4) Secret abstinence societies are well adapted to exercise this extended cure. They not only prevent but lessen the danger of relapse. (5) Temperance abstinence societies should affiliate with public institutions for the treatment of alcoholics. (6) The fact that abstinence can be secured without institutional treatment will make it possible in many cases to shorten the time of treatment now prescribed (from six to twelve months). In spite of these re-

sults he believes that in the majority of cases institutional treatment is necessary. In this treatment, employment of the patients is essential for their betterment, and conduces to economy in their support at the institution.

DEFENDORF (Middleton, Conn.).

ARCHIVES DE NEUROLOGIE

(Vol. XV, 1903, No. 88, April.)

1. Historical and Critical. Physio-psychology of Women in Religious Orders; the Nuns of Port Royal (Fifth Series of Five Observations). CHARLES BINET-SANGLE.
2. Upon Some Clinical Particularities of Facial Neuralgia and Its Treatment by Electricity. A. ZIMMERN.
3. Fibromatose and General Paralysis. DR. A. CULLERRE.

1. *Women in Religious Orders, etc.*—The author presents an interesting study (to be continued) of the lives and mental and physical experiences of three nuns of the famous convent of Port Royal, since destroyed (near Paris, France). The facts are obtained from various historical, biographical and statistical works cited by the author, and the study carries us back to the year 1661, the time of the Jansenist controversy, when the nuns, upon pain of dismissal to other and, in the Catholic sense, orthodox communities, were required to sign a "formulary" condemning certain five propositions contained in Jansen's treatise upon the theology of St. Augustine. When we consider that the propositions were upon such subjects as Free Will, Divine Grace and Predestination, we cannot wonder that the sisters, who had sought rest for their souls behind the convent veil, should have been driven into torment of mind, when compelled to decide whether they were bound in conscience by the decision of the church on those subjects, while in their own understanding, they held different views. Under stress, some would sign, and thereupon be seized with mental torture, dreading the fate promised to liars and hypocrites. Mind acted on body and aggravated physical maladies. The author, after stating at much length the story of fifteen years of bodily illness and of occasional moral suffering, in the life of the nun Margarine Dupr , and relating particularly an account given of her entire relief, in answer to a special prayer, from all her bodily illness for three months, makes the following summary of her case.

Character.—Haughty and of a "nature prompt and lively," she became under the influence of religious suggestions, reserved, quiet and humble. She called herself "a poor ignorant" and busied herself with labors to the most lowly.

Sadness.—She exhibited a predisposition to sadness. In 1661, dysentery declared itself upon an occasion of mental distress. In 1662 her abscesses of the liver were accompanied "with great spiritual suffering." In 1664, during her exile to the "Anonciades of St. Denis," the news that certain nuns of Port Royal had signed the formulary plunged her into dejection. Herself urged by the archbishop of Paris to surrender, "she passed five days in weeping, in despair of her condition, distracted between the fear of offending God in signing and having always to suffer for not signing," "in pain and transported with agony." She finished by submitting and gained only new disquietude. The day of her return to Port Royal, she remained in a corner of the church "praying and weeping." At that time "she slept little at night." Finally, being convinced that the signature was a wrong act, she accused herself of her fault "in terms most humble and most touching and with great abundance of tears." "From that moment she felt the greatest joy that she had ever experienced, mingled with heart-felt grief for her sin." She continued to deplore it and had frightful remorse on account of it.

Suggestibility.—She said *  propos* of the pseudo miraculous, as the

author calls it, remission of her illness: "I have not only received that grace by the intercession of our dear mother, the deceased abbess Mother Angélique, but also some other grace, very secret and very considerable, that I am not able to publish; and I can give assurance that I am never inspired to demand anything of God that I do not obtain it, by her intercession." This gives the measure of her suggestibility. She had made her profession at a convent of the Congregation of Notre-Dame in Flanders, when "at the end of two years the 18 December, 1651, she was received at Port Royal, without *dot*." There she receives the suggestions of Antomé Singlier, director of the nuns and of the abbess Jacqueline Arnaud, who, says she, "takes possession of my heart." Having refused to sign the antijansenist formulary of 1661, she was in 1664 exiled to the Annonciade of Saint-Denis. Here she made nine novines daily. In the first months, she gloried in her resistance, but at the end of ten months of captivity, "she allowed herself to be so carried away by considerations of blind obedience to her superior, that forgetting everything else, she signed the formulary purely and simply under the direction of M. de Pèrefixe," and she did it "with great uneasiness of mind." "She was then ill and feared to die in criminal disobedience." "She signed also the new formulary by the mandate of the archbishop." Returned to Port Royal and again submissive to Jansenist suggestion, she retracted her signature and refused to sign the formulary of 1665. Besides, wishing to multiply the proof of her repentance, she made several copies of her retraction, which she cast from the windows and underneath the walls of the monastery, at that time blockaded. She died at Port Royal in 1666. As to the supposed miraculous answer to prayer, the author considers it either merely a coincidence or that it was produced by "auto-suggestion."

The second observation is upon the case of the nun, Anne-Marie de Flécelles de Brégy, who was a relative of Cardinal de Retz. She had a strength of character which nothing could move. "I am seized," she writes in 1664, when of the age of thirty-one years, "with a certain fear, that I have not enough of fear." And *à propos* of the nuns, who had signed the antijansenist formulary, it has put me in such a trembling and terrible fear lest God should abandon me, that I have instantly supplicated him for grace to cling to him and to suffer all my life humiliations, afflictions and maladies of every sort to obtain the help of his grace and the gift of perseverance never to abandon the truth." "Why shall I not tremble? Why shall I not fear, when we see those fall, by whom one expected the salvation of Israel—It is a sorrow the most affecting in the world."

She was exiled to the Ursulines of Saint Denis, but returned the next year and signed with her blood a declaration that she persisted in her refusal to sign and disavowed all that she should be made to do in her last illness. She resisted to the end, dying at the age of fifty-one.

The third observation is similar in character. The author's conclusions are to follow in later number.

2. *Treatment of Facial Neuralgia*.—This is the conclusion of a previous contribution, relating to the department of Electrotherapy in the Charcot Clinic. The author finds it difficult to explain that certain monographies no longer insist upon the electrical treatment of facial neuralgia and that others pass it by in entire silence. Does not, he says, Gilles de la Tourette affirm that if medical means fail, there still remains surgical treatment, treatment full of dangers and chances, but outside of which there is nothing to try? Does not Mauclair also say that when medical means have been utilized without success, the physician "*ought to give place to the surgeon*," and does not this author counsel to operate at once and without waiting, under the pretext that to wait is to give the nevrite ascendancy and to the encephalic lesions time to evolve? And he adds: "Although the re-

sult should not be perfect (Trousseau affirms that he has never seen a complete cure of tic douloureux of the face), we can say with Monod that one year of well being in the course of an affection which imposes veritable torture constitutes a benefit appreciated by the patient, so much more, as the intervention is free from dangers (nevrectomy)." The author, contrary to this opinion, will not admit that after a conscientious trial of medical methods, the physician effaces himself without hesitation before the surgeon. A place, an important place, he says, should be reserved for electric treatment, which, in all cases, we should have interest to attempt before dreaming of a surgical intervention, however light. The author mentions the various surgical methods and gives some statistics of the great danger of a fatal result in gasserectomy, and in view of the benefit to patient shown in some fifteen cases that he reports at length, he states that he thinks that "course to pursue" should be the following:

(1) In cases of neuralgia, with well determined etiology, we should have recourse to their specific treatment. (2) In neuralgias of a mild type, we should commence by resorting at first to the usual medications, free to recur in case of insuccess to electricity, which will bring about almost certainly a complete cure. (3) In neuralgias of a grave type, after one has established the inefficacy of medical treatments, administered in a methodical fashion (among them opiate treatment) we should submit the patient to *electrization, during a time sufficiently long* to judge of the effects obtained (minimum three months) and we should not decide to employ a surgical intervention, except in case of *absolute insuccess*. In last analysis a peripheric operation might be tried (névrectomy). But it is only *in despair of the case* (en désespoir le cause) and before the formal demand of the patient or the positive threat of suicide, that we should resolve to propose gasserectomy.

3. *Fibromatosis and General Paralysis*.—The author, in presenting the case of a woman afflicted with dermofibromatose, and of whose desperate condition he gives a photograph, asks: Is the affection, known under the name of the Malady of Recklinghausen always congenital? Is it due to an anatomic alteration of the nervous system? It is not yet incontestably established. Still we understand that the authors admit that, in subjects afflicted with this malady, there exists a certain *congenital feebleness* of the nervous system, which places them in a category apart from degenerates. Upon this point, the case we present and which, believe us, offers a case hitherto unique of general paralysis in a woman afflicted with congenital and probably hereditary dermofibromatose, may have a certain interest. The patient was a woman, aged forty-seven, a street vendor, married and with one child; admitted to the hospital in April, 1901; chronic alcoholism, dementia, impulsion to suicide, vague idea of wealth and afflicted with generalized fibromatose of the entire external tegument. The back from the neck to the sacrum is literally covered; upon a foundation of little sessile fibromes are attached numerous soft excrescences with large pedicule, particularly in the median region. The patient affirms that "she had that from birth," and that her mother was afflicted with similar tumors. June 1 she was attacked with erysipelas of the face, following an excoriation of the nose, which later was cured. June 6 an epileptiform attack. In December further series of like attacks. January 5, 1902.—Grippal enteritis of grave form, followed with cachexia, slough of sacrum and progressive marasmus. May 6.—After a series of attacks, the patient, profoundly cachectic, succumbed.

Comments.—We do not know whether, aside from alcoholism, we can attribute any other cause in this case of general paralysis. We are ignorant, notably, whether this patient has had syphilis. The only thing besides which appears to us interesting in this observation, is the coincidence of

fibromatose, an affection congenital and familial, as the patient said (and there is no reason to suspect her veracity, for she has never varied on this point in spite of her state of dementia), and of peri-encephalitis, a malady held as the most accidental of psychopathies, and considered generally as developing ordinarily otherwise than from predisposition and in individuals with brain primitively well constituted. Or, if fibromatose is the index of a *congenital feebleness of the nervous system*, following the exact expression of the authors, this case should be added to those already numerous, where we see general paralysis attack the predisposed, the degenerate, even also imbeciles. In 1888, L. F. Arnaud published two cases of general paralysis in imbeciles, recalled two analogous observations, one of Morel, the other of Christian, and attributed the congenital cerebral feebleness of the patients to the alcoholism of parents. In 1893, my interne, Gagnerot, in his thesis, entitled, "Of Predisposition in General Paralysis," gave some examples of paralysis, grown upon the soil of degeneracy. In 1897, L. Chappelletti published an article upon general paralysis in imbeciles. In 1898, I myself, in the *Annales Medico psycho-logiques*, gave observation of the same kind and advanced the opinion that "almost all general paralytics of the rural class are weaklings." "The observations which I have collected since are not of a nature to make me change my opinion.

RICHARDS (Amityville).

NEUROLOGISCHES CENTRALBLATT

(Vol. 22, 1903, No. 5, March 1.)

1. The Acromial Reflex. W. v. BECHTEREW.
2. The Carpo-metacarpal Reflex. W. v. BECHTEREW.
3. New Contribution to the Physiology of the Tendon Reflexes. Preliminary Contribution. A. E. STCHERBAK.
4. Vesical Incontinence and Paralytic Manifestations in the Extremities in Focal Softening in the Subcortical Ganglion. A. HOMBERGER.
5. Further Contributions upon the Developmental (Myelogenetic) Regions in the Human Cerebral Cortex. P. FLECHSIG.

1. *Acromial Reflex*.—Bechterew calls attention to a periosteal reflex obtained by striking with the percussion hammer, the acromial portion of the scapula and the coracoid process. This reflex consists ordinarily in a flexion of the forearm, sometimes in an inward rotation of the hand and in exaggerated cases in a flexion of the fingers. These movements are produced by a contraction of the cervico-brachialis and biceps muscles. The reflex is obtained in any condition where there is a heightening of the reflexes as in organic hemiplegias and in amyotrophic lateral sclerosis.

2. *Carpo-metacarpal Reflex*.—Bechterew describes a periosteal reflex obtained by striking the carpus and the adjoining metacarpal region resulting in a flexion of the fingers with the exertion of the thumb. The patient's hand should be held with the dorsum upwards. It is obtained in organic lesions situated above the cervical swelling, especially in organic hemiparesis or hemiplegia, as of cortical or capsular origin.

3. *Physiology of Tendon Reflexes*.—Stcherbak records some interesting experiments upon animals. By means of a tuning fork rapid vibrations to the hind leg of rabbits were applied, which resulted in a heightening of the knee reflex, knee clonus, which could be produced by tapping, and passive movement of the knee joint and spastic tremor. These phenomena could be produced by irritation of the other foot. Rapid passive movement 1000 to 1500, also produces a similar condition. Cutting the spinal cord above the center for the knee reflex and applying rapid vibration produced a heightening of the knee reflex, but no clonus or spastic tremor. These vibrations had no influence upon the animal's general condition or muscle

tone. Vibrations applied to the spine, especially the lower dorsal region, produced a spasticity of all the muscles of both hind extremities. Stcherbak concludes as a result of these experiments that by means of vibration we can so influence the lower, somatic portion of the nervous system, that phenomena are produced which we usually ascribe to the higher or psychical apparatus, and that these phenomena may be latent for some time afterwards.

4. *Subcortical Ganglion*.—Homberger describes the symptoms of ten cases in which numerous minute areas of softening were found in the corpus striatum and thalamus. In six of the cases the lesions were bilateral. Incontinence of urine was present in all the cases. Micturition was of the automatic type. There was partial paresis of the lower extremities, and increase of the reflex. The upper extremities were also somewhat affected. Forced laughing and crying and occasional hemichorea and hemi-athetosis were present. Homberger concludes the following as the result of lesions in the corpus striatum and optic thalamus: (1) Unilateral lesions cause transient incontinence and a permanent increased desire for urination; (2) bilateral lesions cause permanent incontinence of urine, which does not differ from the spinal form; (3) the subcortical innervation of the bladder is bilateral; (4) superficial lesions do not cause incontinence; (5) bilateral areas of softening in the basal ganglia cause disturbance of station and paralysis which differ from those cases where the lesions involve the cortical fibers of the internal capsule.

5. *The Developmental Areas of the Cortex*.—Flechsig gives the result of his study of six more brains with regard to the date of the appearance of medullary sheaths in the various tracts of fibers proceeding from or to the cortex. This makes a total of 52 brains which he has examined. He modifies slightly his previous statements: (1) Flechsig divides the cortex into 36 regions. He adds a new area to the first parietal convolution, making three areas. This region is found near the intraparietal nucleus and resembles the subangular convolution in size, the arrangement of its fibers and the date of appearance of the myelin sheaths. He is not certain that fibers from the corona radiata enter this region. A new area is distinguished in the upper portion of the occipital convolution, making three areas in the sight region; (2) regarding the order of development the following is of importance. The motor convolutions show the most marked development in the more fully developed fetuses. In a brain of a 34 c.m. long fetus he found a well developed bundle of fibers which were traced to the olfactory sphere, while the fibers of the cerebral convolutions were not differentiated. Unless this is an exceptional case, the olfactory sphere should be designated No. I, and the central convolution as No. II; (3) it is possible to distinguish between the motor and sensory bundles of fibers. The motor fibers are found anterior to the central fissure and the sensory fibers posterior to this. In the supra and subangular convolutions the types are not distinct: (4) in 1901 Flechsig demonstrated that to every sensory (centripetal) bundle there corresponds a motor one. He considers now that the projective system of the brain cortex is composed of similar conjugate pairs of bundles; (5) the order of development of the brain sulci has an important bearing upon the order of development of the myelin sheaths. The areas indicated as primary appear very early while the sulci termed as terminal appear late. This is not definitely proven, but the relation is established.

(Vol. 22, 1903, No. 6, March 16.)

1. Contraction of the Sphincter Iridis in Pupils Immobile to Light, in Accommodation and Convergence Reaction. M. ROTHMAN.
2. Anatomical Changes After Crushing the Roots of the Spinal Cord in Dogs. S. BICKELES.

3. Neurotonic Pupil Reactions. J. PILTZ.

1. *Pupillary Changes*.—Rothman reports a case of a girl twelve and one-half years of age who had occasional periods of unconsciousness during childhood. At five years of age she developed periodical attacks of migraine. After one of these headaches it was noticed that the right pupil was dilated at maximum and was absolutely immobile. The left pupil was normal. Potassium iodide was given. After a period of five months there was a slight reaction of the pupil in accommodation and convergence. During the whole period of observation, three and one-half years, no reaction to light was observed. In the course of time the right pupil regained its normal size, but would remain contracted 30 to 40 seconds after accommodation and convergence. The case represents an isolated paralysis of the right sphincter iridis, the lesion probably being a small hemorrhage in the oculomotor nucleus.

2. *Crushing the Spinal Roots*.—Bickeles crushed the anterior and posterior spinal roots of dogs either with pincers or ligatures for five minutes. The dogs were killed from seven weeks to two and one-half months after operation. Examination showed in the posterior roots a large number of fine regenerated fibers, the number depending upon the length of time elapsing after the operation. Bickeles also found what he believed to be regenerated nerve fibers in the intramedullary portion of the compressed posterior roots. This, however, was not very active, probably due to the increase of neuroglia tissue and a sclerosis. Examination of the posterior roots of dogs who died two to two and one-half weeks after operation showed an absence of centrifugal fibers. In the anterior roots a band of fibers was found which were traced as coming from the posterior roots. Degeneration was always on the same side as the injury.

3. *Neurotonic Pupil Reactions*.—Piltz discusses the importance of pupillary phenomena. In 37 cases of progressive paralysis, 11 did not respond to light on either side; 10 in whom the light reaction failed in one and was poor in the other; 7 in whom the reaction was poor in both sides; 4 in which only one pupil reacted poorly and 5 in whom the light reaction was normal. In 63 cases of tabes, in 37, both pupils failed to react to light; in 6 the pupils on one side failed, the other being slow; in 15 both pupils reacted slowly; in one, the pupil on one side reacted poorly, the other normally, and in 2 cases the light reactions were normal. Impairment and reaction manifests itself either that the pupil does not contract as much as it should or that it does not contract as rapidly as normally. It also manifests itself in a persistence of the narrowing of the pupil for some time after reaction. Four cases are recorded with the later phenomenon.

WEISENBURG (Philadelphia).

MISCELLANY

A NOTE ON TRAUMATIC SYRINGOMYELIA. Alfred Gordon (Philadelphia Medical Journal, May 9, 1903).

The writer reports a case of a woman aged forty years who, ten years before, had slipped and fallen on her back. This was followed by numbness and a sensation of pins and needles, and burning and aching in both hands. The pain disappeared but the numbness persisted. The hand would not be pained by burning, though covered by blisters. Three years ago a red swelling on the palm of the hand developed, which increased. A subsequent dislocation of the arm was painless. The motor power in both lower and right upper extremities was normal. The left arm was weaker and larger than the right, each finger being larger than the corresponding finger of the right hand. The subcutaneous tissue seemed hypertrophied. No hysterical stigmata were noted. The skin was soft and thickened. The reflexes and reaction to Farradism were diminished on the left arm.

Anesthesia of side of face and head was present. Temperature sense (heat and cold) were lost over the whole left arm and half the thorax to sternum and the middle line of the spine. Loss of pain sense was identical with thermo-anesthesia. Very little muscular wasting was present. The case was evidently a typical sensory dissociation characteristic of syringomyelia, also trophic changes of the tissue underlying the epidermis. The cause was probably the traumatism causing a hemorrhagic focus, developing into a gliosis or cavity of the spinal cord. Trophic disturbance in syringomyelia of the nature of edematous swelling of paralyzed limbs are not rare. A hard edema due to hyperplastic process in the subcutaneous tissue was described by Marinesco under the name "main succulente." The vasomotor and trophic centers for the bones and integument are situated in the gray matter of the posterior cornua, about the eighth cervical.

NOYES (New York).

DO OUR PRESENT WAYS OF LIVING TEND TO THE INCREASE OF CERTAIN FORMS OF NERVOUS AND MENTAL DISORDER? Charles E. Atwood (New York Medical Journal, June 13, 1903).

Insanity is increasing slightly, especially degenerative types. Neurasthenia is increasing. This increase is found mostly in the immigrant population, 75 per cent of the insane of the State and 80 per cent of the cases of neurasthenia at the Vanderbilt Clinic being either foreign born or of foreign parentage.

Our present methods of living entail an increased mental strain, the number of sensory impressions and variety of ideas forced upon us by our increasing interests permit scarcely time for the reception of impressions, and no time for their proper assimilation. Emotional stresses, e.g., those caused by sudden and great reverses of fortune; profound impressions induced by fanatical doctrines, impressions induced by certain lurid daily newspaper are deleterious to the neurotic. Over indulgence in eating and drinking tends to arterial sclerosis. Tendency to auto-infection is assisted by irregular living, and auto-infection is a recognized cause of certain forms of insanity and certain neuroses. The immigrant suffers the most on account of competition with a superior race, deficient alimentation and resultant anemia, other concomitants of poverty, and faulty hygienic conditions. The consumption of alcohol per capita in this country has increased, and our women are drinking more and more each year. This has a tendency to establish a faulty heredity and induce a strong predisposition to nervous disorders.

AUTHOR'S ABSTRACT.

BRACHIAL PARALYSIS—POST NARCOTIC. F. J. Cotton and S. W. Allen (Boston Medical Journal, May 7, 1903).

The writers report four cases of post-anesthetic paralysis, and collect thirty from general literature. The patient is anasthetical with the arms drawn up over the head, and after recovery one or both arms are found paralyzed. There is little or no pain and slight sensory disturbance. The muscles of the shoulder and arms are affected in a varying degree—sometimes all are involved; sometimes the shoulder is spared, and the hand and arm muscles are alone affected. In a few days there is beginning of return of function, more usually in the hand, then a slower return of power in the extensors to the deltoid and to the rotators of the shoulder. As the function returns many of the cases present a paralysis approximately of the "Erb" type (deltoid, biceps, brachialis anticus and supinator longus). In a very few cases there is involvement of the sympathetic root communicating with the first dorsal root, signified by pupillary and other ocular changes. Later, soreness of the nerve trunks and of the muscles may appear. Reflexes are usually normal. Electrical tests rarely show a partial reaction of degeneration; usual reactions are normal. There are three

possible mechanisms by which these paralyses may occur: (1) Pressure on the plexus, (a) exerted between the clavicle and transverse processes of the vertebræ, and (b) between the clavicle and the first rib; (2) tension on the roots or plexus from the position of the arm; (3) tension from position of the head or neck, the arm being abducted.

NOYES, New York.

IMPERATIVE CONCEPTIONS. L. Harrison Mettler (Medical Record, April 4, 1903).

Imperative conceptions or ideas are those involuntary, unrelated persistent notions that spring up in the minds of many victims of nervous disease. Though found in many states of mental and physical debility, they belong essentially and entirely to the psychoses. A sharp distinction should be made between the imperative conceptions of the sane and the insane. In the sane the person's judgment remains unaltered; he tries to resist them and often succeeds. The imperative conceptions of the insane become for the time being the patient's very ego. The insane delusion is deeper and more influential than the imperative conception, entering into the constitution of his ego, and coloring and outlining his objective world. The so-called imperative idea, on the other hand, is recognized by the patient to be a mere subjective phenomenon, annoying and persistent though it may be. It may involve all the faculties of the mind, not alone the imagination, but there are usually no distinct hallucinations or delusions with these impulsive conceptions. Insane delusions are, primarily, the result of a diseased cortex; imperative conceptions are the result generally of disease outside the brain.

NOYES (New York).

NEURITIS FROM WHOOPING COUGH. Charles E. Aldrich (N. Y. Medical Journal, June 6, 1903).

Neuritis may attend or follow any of the acute infections, especially those characterized by prolonged high fever, marked disturbance of nutrition and great prostration. The author reports a case of a child aged four, who, after whooping-cough, developed weakness, pain in the legs, weakness of grasp of hands, nasal speech and regurgitation of fluids from the nose, diminution of knee jerks and loss of touch and pain sense in the hands and feet. Recovery came after twelve weeks. Various forms of paralysis have been observed to complicate or follow whooping-cough. Literature proves that it is rare. Sixty-two cases reported were cerebral in origin. Several cases of general peripheral neuritis were collected from literature.

W. B. NOYES, New York.

DEVELOPMENT AND CARE OF CHILDREN. J. C. Cook (Journal of the American Medical Association, June 6, 1903).

The changes in the nervous system from birth to maturity afford us many examples of the differences between the child and adult. At birth the central nervous system, as well as the peripheral, is almost entirely unmyelinated. Hence we find the fibers of the spinal cord, the medulla, corpus quadrigeminus and the pons varolii mainly somatic. The motor nerves receive their attachments much earlier than the inhibitory centers are developed, hence the objectless movement that characterizes the unmeasured exercise of the young. Thus maturity is gradual, and full growth is only reached between puberty and adult life. It is our duty to guard and conserve every atom of energy through this period of unfolding and budding life, realizing that they are bundles of possibilities unsolidified, which begin at the beginning and end we know not where. The study of biology teaches that the higher the organism, the slower the development, hence the necessity of a long period of protection and care. Take with this the long periods of stress and functional demands on the nervous system and

we can realize the greater necessity for a wise and careful guardianship. The writer urges strict control of the question of child labor, and advises a commission of five members, at least two of whom shall be physicians, to have jurisdiction over all educational and labor questions.

W. B. NOYES, New York.

EFFECTS OF THE STREPTOCOCCUS ON THE CORTICAL NERVE CELL IN MENINGITIS. Leonard K. Hirshberg (Maryland Medical Journal, July, 1903).

Autopsy: Male, thirty-nine years. Clinical diagnosis: Fractured skull, cerebro-spinal meningitis, myocarditis, endocarditis, pyemia, pneumonia at base of right lung. Anatomical diagnosis: Purulent traumatic cerebro-spinal meningitis, depressed fracture of skull; rupture of dura, depression and bruising of right cortex; fatty liver. Brain: Subarachnoid space contains exudate, consisting of two zones. Inner zone, in contact with cortex, consists of network of fibers, including many cells with long vesicular nuclei, surrounded by much less staining cytoplasm. Probably proliferated endothelial cells from serous surfaces and lymph spaces of membrane. Few poly morphs nuclear leucocytes in the zone. Outer zone consists, almost, of poly morphs nuclear leucocytes. Tissues of pia richly infiltrated and thickened by exudate of pus cells, in which numerous proliferated endothelial cells are made out. Weigert's method shows moderate number of streptococci in exudate. Cortical sections, from area of Rolando, stained by Nissl, show disappearance of tigroid bodies from cells. Many cells also exhibit loss of nuclei. Cells shrunken and distorted where nuclei remains. Nuclei swollen, vesicular, limiting membrane at times in contact with body wall of cell. Eccentricity frequent as to nuclei and nucleoli. Direct or immediate action of the toxin of the streptococcus seem probable.

J. E. CLARK, New York.

THE EPILEPTIC AURA. William P. Spratling (Medical News, July 18, 1903).

Four types of auras are studied: (1) Psychic, (2) sensory, (3) motor, (4) irregular. In a close analysis of 815 males and 510 females, 36 per cent were found to have a sensory aura of some sort, 4 per cent had a psychic aura, and 2 per cent a motor aura. Of the entire number, 45 per cent had some aura, 55 per cent none. The sensory auras are not only common, but extremely varied, affecting any of the special senses, including marked perversions of the same, and being brief or rather prolonged. Visual auras predominate, and appear either as flashes of light or colors, or optical delusions, or temporary blindness. The epigastric aura appears in 15 per cent of all cases, and is the most frequent. The problem in epilepsy is to determine whether the initial disturbance, the thing that breaks up the rhythmic periodicity of the respiratory impulse, is to be sought in the respiratory center primarily, or whether it lies somewhere in the periphery, in the stomach, or elsewhere, and represent the original source of the epilepsy. For instance, when a case with epigastric aura suffers from periodic indigestion, gastric catarrh or flatulence, the probability is that the same cause makes the aura and the flatulence, and there is some subtle agent in the unknown field of chemical pathology.

W. B. NOYES, New York.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

THREE CASES OF TUMOR INVOLVING THE SPINAL CORD;
TREATED BY OPERATION.¹

BY DR. JAMES J. PUTNAM

AND

DR. J. W. ELLIOTT

These cases are reported in order to show the benefit that may result from operations for tumors involving the spinal cord, even when it is not possible to effect a complete cure; and also to illustrate the danger with which such operations are attended.

In the first case to be described the result was favorable to a degree far exceeding our expectations. It may be that the operation was only partially responsible for the eventual improvement, but it cannot possibly be doubted that it furnished the controlling factor.

Case 1. The patient, who was a young man, twenty-three years old, in the employ of the West End Railroad Company, was brought to Dr. Putnam in December, 1897, and gave the following history:

He said that, just three years before, he was doing a job of work which required him to mount on one of the tall ladders reaching to the trolley wire, and that while descending he missed

¹Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

his hold and fell. He grasped the ladder as he went down and in that way broke his fall, but at the same time his neck received a sudden twist. He picked himself up without help and walked a distance of about a quarter of a mile, suffering only from pain in the neck; and then took a car home.

That night he suffered from pains in the neck, and for two or three weeks afterward he stayed in the house, not feeling able to work. There was no disturbance of motion or sensation of any kind, except that his head was held somewhat askew, and that attempts to turn it to the left caused pain in the neck.

Six weeks after the accident he went to work again, and worked for another year, feeling well, but not able to turn his head quite freely from side to side. Then he again received some slight injury, in consequence of which the neck once more became actively troublesome for a time.

In December, 1896, that is, a year before the first examination was made, he was off work for two weeks; then he worked again until April, 1897, when he gave up for good and all, on account of renewed pain in the neck.

In the following month, that is, in May, 1897, he fell while running—perhaps because he tripped, perhaps because new symptoms were coming on—and on this occasion also the neck was painfully jarred.

From that time on he was unable to run, though for several months more he could walk, he thinks, nearly as well as ever.

The evidence up to this point makes it clear that the original injury was the primary factor in setting up the subsequent morbid process, and this conclusion seems the more justified from the fact that he was never able to turn his head freely after his first accident, and less well to the left than to the right.

In July, 1897, his gait became awkward, and in August his hands began to feel numb and would shake on use.

For several weeks before the first examination his back and sides had been feeling weak, and breathing had been getting more and more difficult. There had been no paralysis of the sphincters.

The physical examination made on December 27, 1897, showed that the movements of both arms were weak, and that the difficulty of motion increased distally. Thus, his fingers were semi-flexed, and further flexion or extension was impossible; flexion and extension at the elbow were possible, but very feeble; abduction at the shoulder was better but also feeble, and not so good on the right side as on the left. Passive extension-movements of the fingers excited strong clonus.

The prick of a pin was felt fairly well on both hands, but, unlike the motion, it was better for the left side than for the right. [Brown-Séquard complex.] Light contact was felt on both hands

and arms, and this form of sensibility seemed to be less affected than that of pain. The movements of the legs were also weak, but were all possible. The right leg was stronger than the left. The knee-jerks were greatly exaggerated, and ankle clonus was present. The respiration was labored, and the accessory muscles of inspiration were called strongly into play. The temperature, by the mouth, was 99.7° F. On palpation in the pharynx the bodies of the upper cervical vertebræ seemed to be unusually prominent.

The presence of this group of signs—the involvement of all four limbs, of all the costal muscles, and even of the diaphragm—made it obvious that the patient was suffering from a lesion high in the cervical region, and a lesion of such a character as to affect, in some measure, the whole thickness of the cord, the centripetal as well as the centrifugal tracts. The whole course of the disease, the history of neck-injury resulting perhaps in partial dislocation of the cervical vertebræ, the prominence of the upper cervical bodies in the pharynx, all pointed to an affection of the bony structures, while the high exaggeration of the wrist-jerks and knee-jerks bore out the idea that the nutrition of the cord was being impaired rather by pressure, with its attendant circulation disturbances, than by a destructive lesion primarily involving the neural tissues themselves.

It was thought at first that the bone disease was tuberculous, but Dr. Walton, who made an examination at a later period, favored the diagnosis of a sarcomatous growth at the seat of injury, and this proved to be correct.

The patient was at once referred to the Massachusetts General Hospital, which he entered on December 28, 1897, coming under the care of Dr. F. C. Shattuck. At entrance his temperature was 100.2° F., pulse 112, respiration 30. The thoracic and abdominal organs were normal. The arms were constantly held semi-flexed and the fingers nearly closed, but he was able to walk up and down stairs with the aid of one man.

After staying a few days, the patient was discharged at his own request, but soon found his legs getting weaker and his breathing more labored, though he did not lose weight.

A few months later he was again seen by Dr. Putnam, and operation was advised, at least for exploration. With this in view he re-entered the hospital on June 19. At this period his fingers were immovable, and his arms and legs utterly helpless, though not absolutely paralyzed. Micturition was voluntary, but the movements of the bowels were secured with difficulty. The tendon reflexes were highly exaggerated, as before.

The head was partly extended, and could be moved to about three-quarters of the normal extent, in all directions; a resistance could be felt deep on the left side of the neck, at the level of the

second and third cervical vertebræ. A prominence corresponding to the body of the second cervical vertebra was to be felt in the pharynx.

An attack of difficult breathing precipitated the operation, which was made on June 20, the day after his entrance.

Operation. The patient was placed face down, with his head bent sharply forward over the end of the operating table. An incision was made from the occipital protuberance down in the median line nearly to the seventh cervical spinous process. The first, second and third vertebræ were exposed and their laminæ removed. The posterior surface of the axis was found to be eroded, so that on grasping it with forceps it came away as a thin shell of disintegrated bone, disclosing a small grayish lobulated tumor, the size of an acorn. This had every appearance of a sarcoma and was removed with scissors and forceps. It seemed to involve the dura and perhaps the cord itself. The base was curetted, and the curette followed the disease around into the body of the second vertebra, care being taken not to injure the vertebral artery. A quantity of the disease was then removed from the vertebra in front of the cord. Horsley's spine forceps were found to be admirable for cutting the laminæ in this very deep region. The operation was difficult and severe.

The wound was closed without drainage, and the patient made a good recovery from the operation. His respiration at once became much easier than before the operation. There was no pain, and the wound healed by first intention. Within three days the patient discovered that he could move his legs better than before, flex and extend his right hand at the wrist, and move his thumb and fingers.

The pathological report on the specimen removed was as follows:

"An irregular mass of tissue with some spiculæ of bone on it. Microscopic examination showed solid masses of irregular round cells interspersed with large multinucleated ones. Giant-cell sarcoma."—Wm. Whitney, Pathologist.

On June 29 he could flex both forearms on arms to a right angle, and could also flex both thighs. All voluntary motions were much increased over what was possible before the operation. Improvement was reported in movements of arms, legs and neck. Respiration was quiet and natural. July 2, slow but steady improvement was reported in movement of arms, legs and neck.

July 11 he was discharged from the hospital, "relieved."

On April 18, 1899, the patient was re-examined at his house and gave the following report:

He left the hospital a good deal relieved, he said. After getting home he at first lost a little, but then improved again so that,

with assistance, he could move about the room a little. He could also lie on his side, in bed, which before the operation had been impossible. After this he grew worse once more and was pretty poorly for several months, although he never lost all that he had gained.

In October, that is, three months after leaving the hospital, the improvement recommenced, and from that time on it had continued without interruption.

At the time of this examination he could raise his hands freely, though rather awkwardly, to the head and above the shoulder, could feed himself with the left hand, and could hold a book and turn the leaves. His fingers were stiff and weak, especially the little finger, but all movements of them were possible, whereas before the operation they were wholly incapable of motion. The legs were stiff, but here also all movements were possible, even those of the toes, though the attempt to move them would bring on extension clonus. He was not able to walk wholly alone. He could turn the head freely to the right, and could move it forward and back, but could not turn it much to the left. The breathing gave him but little trouble, and was both costal and diaphragmatic. He had no pain. His flesh and color had improved. The bowels moved freely and the micturition was normal. The knee-jerks were very markedly exaggerated, as well as all the deep reflexes of the four limbs. His sensibility to contact, pricking and changes of temperature was almost, if not quite, perfect over all parts of the body below the head. The sense of position was good. The improvement continued, so that on the following Thanksgiving Day he was able to walk downstairs and take part in the family dinner.

A final examination was made on January 2 of the present year, 1903, with the following results:

The patient reports that he had been steadily gaining, and that he is now keeping a small store entirely alone. His physical condition is as follows:

The gait is very spastic. On turning round he loses his balance easily, and he sways on attempting to stand with eyes closed. There is no ataxia of the arms, and the grasp of both hands is strong. The wrist and elbow jerks are markedly exaggerated, but there is no muscular atrophy of the hands. The knee-jerks are very lively on both sides, and ankle clonus is present. There is no disturbance of sensibility of the hands or face. The pupils are of moderate size and react normally. The pulse and respiration are both normal. All movements of the head are apparently normal, except that of rotation, which seems somewhat restricted. His neck looks short, as if his head had settled somewhat between his shoulders. There is no sign of recurrence of the new growth.

This case is especially interesting in two respects: (1) Although new growths of the cervical region have several times been reported, this is the first instance in which an operation has been made for a tumor at this level, the one which comes nearest to it being that which is described in the recent and interesting report by James W. Putnam, William C. Krauss and Roswell Park,² when a sarcoma was successfully removed from the neighborhood of the fourth and fifth cervical segment. (2) It is important as showing how much can be accomplished even where the disease with which the surgeon has to deal is of the degree of malignancy that belongs to the sarcomas; and how long the tendency to recurrence may be delayed. It was equally true of the case described by J. W. Putnam, Krauss, and Park, that the tumor was sarcomatous, and the symptoms were equally serious with those exhibited by our patient, the disorder of sensibility, indeed, even greater; yet here, also, improvement seems to be still going on after an interval of eight months.

Case 2. This patient was a woman, Armenian by birth, married, thirty-two years old. She was first seen by Dr. Putnam in February, 1902, on account of intense pain through the body and thighs, increased by the slightest movement, and for muscular weakness, which, together with the pain, kept her immovably confined to bed.

The preliminary examination seemed to justify the diagnosis of new growth, probably carcinoma, involving the spinal cord, and on this account she was referred to the Massachusetts General Hospital, where she came under the care of Dr. Elliot. The following notes are taken from the surgical records:

It appeared that twenty-one months before her entrance to the hospital she had been operated on in Worcester for a cancer of the breast, the tumor having been developing for nine months previous to operation. About a year after this she was shaken up in a street railway accident, and one week later she began to feel shooting pains in the sacral region. Shortly afterwards the lumbar region and both flanks became involved, then the left leg, and finally the right leg. In the legs the pain mainly followed the course of the sciatic nerves. So far as she knows, the sensibility of the skin and the muscular power were at that time unimpaired. The pain was always worse on the left side than on the right, and when severe it radiated to the front of the abdomen. From this

² American Journal of the Medical Sciences, Jan., 1903.

time she grew steadily worse and became eventually unable to walk, so that for four or five months she had been in bed. Every movement increased the pain, and recently it had become so distressing that she could not be moved in the slightest degree without extreme suffering.

The physical examination showed the heart and lungs to be normal, and the abdominal organs also normal. The muscles of the legs were flabby, but not markedly atrophied. There was no edema anywhere, and no tenderness on pressure over the nerve trunks. An examination of the back showed a small knuckle of protruding bone involving the spines of the last dorsal and the two upper lumbar vertebræ.

While at the hospital she had poor nights, sleeping but a few hours at a stretch, and suffered from intense, cramplike pains in the thighs after resting in one position for any length of time. Several one eighth grain doses of morphia were required every night to secure even tolerable comfort.

The original diagnosis having been confirmed, operation was decided upon and was performed as follows:

An incision ten inches long was made over the lower dorsal and upper lumbar region, and the laminæ of the last dorsal and two first lumbar vertebræ were removed. On opening the canal a new growth, of carcinomatous appearance, was found presenting immediately beneath the bone. It surrounded the nerve roots on either side of the cord, and extended into the tissues outside the vertebræ. This was removed so far as possible by excision and curetting, and two nerve roots on each side, which were at the level of the growth, were divided, inside the spinal canal. The dura was not opened.

In the afternoon the patient suffered from shock, but revived under the use of intravenous infusions and strychnine. During the next few days she gained strength and was fairly comfortable when at rest, though suffering greatly when moved. Her condition varied greatly, and on some days she suffered continuously. The wound healed by first intention. Three weeks later she was reported as looking well and suffering very little pain, but with considerable paresthesia in the limbs. One week afterward, that is, on March 22, she could move the legs more freely, without pain, and suffered but little pain on being moved by the nurse. The improvement in these respects was so great that on March 26, when she slipped out of her chair one day by accident, very little pain was excited. On April 4, that is, nearly a month after the operation, she was reported as feeling well except for slight dragging sensations in the right thigh. On April 7 she was discharged much relieved, and it was felt that the purpose for which the operation had been made, namely, the relief of pain, had been

accomplished so far as could possibly have been expected.

The plan of dividing posterior nerve roots had been formed before the operation was begun, and it was even felt that, if no other course offered itself, it might be justifiable to divide the cord above the new growth, so imperative was the need of relieving the extreme suffering.

On leaving the hospital it was found possible to transport her to her home in Worcester, and here she remained living in comparative comfort, still suffering much pain, but not of the same severity as before the operation.

In June a convulsion occurred, followed soon afterward by a second. This seemed to indicate the development of malignant growth in the brain, and within a few weeks from the appearance of these symptoms the patient died.

The best indication of the value of the operation is afforded by the recognition on the part of the patient and her husband that the excessive severity of the pain had been considerably tempered. Indeed, the testimony increases that we should not wait for the assurance of absolute success in the case of either cerebral or spinal tumors. The notable improvement recorded by Dr. J. J. Thomas,³ from operation on a myeloma of the spine with compression of the cord, is another illustration of the justice of this view.

Case 3. This patient was a man of fifty-four, married, of good previous health, who entered the Massachusetts General Hospital in the service of Dr. F. C. Shattuck, on January 5, 1902.

The hospital records state that five years before, while suffering under the strain of excessive work, he began drinking champagne, and then whiskey, and he finally took up to the amount of half a pint or a pint a day. Three months previous to his entrance he stopped this habit absolutely. He had never had specific disease. Seven years before entrance he had an attack of what was called rheumatism, in the muscles of both sides of the trunk, and was then in bed eight or nine days, suffering intense pain. Since then he had had occasional attacks of a similar sort, but less severe. The pain was sharp and lancinating in character, but did not entirely encircle the body. Two years before entrance he awoke one morning with queer feelings in his head and left half of his body. There was no loss of power, but a sense of numbness and more or less impairment of cutaneous sensibility, accompanied by dizziness. After a few moments he

³Boston Medical and Surgical Journal, 1901, Vol. 145, p. 367.

seemed perfectly well again, except that the dizziness persisted for some time. Six weeks later, while at work in his office, he again became dizzy and faint, and this time he lost power in his left side and had a recurrence of numbness, together with tingling sensations. These signs were most marked in the arm. After a few hours his muscular power began to return, although the left arm never fully regained its strength and afterward remained subject to numbness and tingling. Six months before his entrance he began to suffer from pains of intense, radiating character, beginning in the back of the neck and extending through the left shoulder and arm. In the morning on waking he often used to find his head drawn to the left side. The muscles of the hands gradually became weak and atrophic, so that the fingers could not be used for any delicate work; also considerable cutaneous hyperesthesia developed. Two or three months later there was some involvement of the other arm. In spite of the intense pain, he attended to his business regularly. About two months before entrance he began to suffer from a painful girdling sensation from the level of the ensiform cartilage to the groin.

There was no pain in the legs, but they would often shake violently. There was no disturbance of micturition, nor any loss of control over the rectal sphincter. These symptoms were soon followed by a loss of power of movement of the legs, which lasted for three weeks, though it finally passed away to a great extent. The muscles of the legs grew wasted at this period in some degree. No mental symptoms occurred at any time, nor any headache or vomiting. At times the voice was noticed to be a little husky.

An examination of the blood showed Hgb. 95 per cent; leucocytes 9800.

The physical examination at the time of his entrance into the hospital showed his thoracic and abdominal organs to be negative, and his general condition of nutrition normal. The wrist reflexes were also normal, but the knee-jerks much increased. The Babinski reflex was present on both sides, but most marked on the right. The cremasteric reflex was normal. There was no disturbance of sensibility of the skin of the head and neck. All the muscles of the hands and arms were more or less atrophied, more especially those of the left arm. This atrophy was most marked in the interossei. No hyperesthesia could be made out at any level, but slight stimulations would frequently excite spasms of pain across the shoulders. The grip was weak on both sides, more so on the left. The breathing was almost exclusively diaphragmatic.

An examination of the cutaneous sensibilities of the chest and abdomen showed an interesting dissociation of the sensory func-

tions of the skin. Thus, from the level of the nipple in front and spine of the scapula to the crest of the ilium, behind, and to just above the pubes, in front, there was an almost entire loss of the sensibility to heat and cold, pain, space, and pressure, while the sense of contact in this area was nearly normal. The cutaneous sensibility of the legs was abolished.

The muscles of the legs were almost completely paralyzed, and toe-drop was marked, but atrophy was not present to any extent. Reflex spasms were frequently induced by slight stimulation of the feet.

Massage gave great relief, and the patient used to rest quietly after it.

On January 7th, the patient was examined by Dr. Putnam and Dr. Taylor (also afterward by Dr. Walton), who reported that the signs indicated disease in the lower cervical region, which was primarily external to the cord; that is, either a cervical pachymeningitis or a new growth. It was also thought to be conceivable that the case might be an unusual one of syringomyelia. It was believed that hemiplegic symptoms pointed probably to an independent vascular disease of the brain, unless possibly both might have been due to multiple sarcomata.

Operation was recommended, though the outlook was not considered as promising. It was believed that the lesion must extend high enough to affect the nerves of the fifth or sixth cervical segment, and that it was most severe at the level of the seventh segment. The fact that so much pain was present and was increased by motion, made it probable that the lesion was meningeal, at least in part. The prognosis without operation was considered poor, especially in view of the involving of respiration.

Potassium iodide having been given in large doses without relief and, moreover, being constantly required to relieve the pain, the patient was transferred on February 4th, to the East Surgical Service, and was operated upon, on February 7th, by Dr. Elliot, as follows:

An incision ten inches long was made from the base of the head to between the shoulder blades, and the laminæ of the first dorsal and the four lower cervical vertebræ were removed. As soon as this was done, a new-growth, of grayish translucent appearance, was found encircling the cord and dura like a collar. It occupied the posterior and left lateral part of the extradural space, and was adherent to the dura, though easily stripped from it. This growth had extended longitudinally for a distance of three or four inches, and had spread into the various openings between the vertebræ. A long and careful dissection was made, for the purpose of removing all visible disease. The dura was

not opened except at one point, where the growth was found adherent, but from this opening a large amount of cerebrospinal fluid escaped.

The operation was a severe one, and at times the respiration was very poor—partly, perhaps, on account of the patient's position. He reacted well, however, to stimulation afterwards, and slept quietly in the afternoon with the aid of morphine.

The next day he was found to be in great pain, with a temperature of 106 F. and with very rapid breathing. On the second day, February 9th, the temperature had risen to 108 F. The breathing was still more labored, and the patient was semi-conscious. His pulse gradually became weaker, and he died about 2 P. M.

In spite of this unfavorable result, Dr. Elliot felt that the position of the growth and the fact that it was possible to remove it almost wholly without any serious injury to the spinal cord made the operation appear thoroughly justifiable, and that in another similar case the good result which was anticipated might readily follow.

The report of the pathologist, Dr. J. H. Wright, was as follows:

"East Surgical. Stevens. Service of Dr. Elliot. Feb. 7, 1902. Laminectomy.

"The material received for examination from the operation consists of several pieces of grayish translucent homogeneous fairly firm tissue and the laminæ or parts of laminæ of three vertebræ.

"This tissue is intimately adherent to parts of the posterior ligaments belonging to these laminæ.

Microscopical examination of sections from the tissue show that it is composed of small cells and coarse fibered connective tissue. The cells have little or no cytoplasm and seem to consist of nothing more than nuclei which are generally rounded in outline and generally about the size of lymphoid cells. The nuclei are also present. In some places the cells are scattered among the connective tissue fibers. In other places there is little connective tissue and the tissue seems to be made up largely of the small cells. Some capillary blood vessels are present.

"Diagnosis: Fibrosarcoma, probably originating in the posterior ligaments of vertebræ.

"J. H. WRIGHT."

This case thoroughly recalls that reported by Henschen,⁴ of Upsala, and Lennander, by whom the operation was performed.

⁴Cited at considerable length in the *Neurological Jahresbericht* of Mendel and Jacobson; for 1901. 5th Jahresbericht, p. 507.

Here, also, a similar congeries of symptoms to those which our case presented was held to point to a growth between the 5th cervical and the 1st dorsal segment, and in fact a spindle-shaped, flat tumor was found. It proved to be non-malignant in character and complete recovery followed its removal. It cannot be doubted that our patient would at least have improved greatly, had the operation not been followed by the rapidly fatal symptoms of the first days. The cause of these symptoms is not clear, though this occurrence after opening of the spinal canal is unfortunately, not unfamiliar.

Finally, this occasion may be taken for reporting that the patient from whose spinal canal a fibroma was removed by Dr. J. C. Warren in 1898,⁵ has continued to improve steadily and is now comparatively free from annoying symptoms.

A summary of the published accounts of operation for tumors of the spinal region has been so recently given in the paper by J. W. Putnam, Krauss and Park, that it is unnecessary to give further details in this place.

⁵Report by Drs. J. J. Putnam and J. C. Warren; *Amer. Journ. of Med. Sciences.* Oct. 1899.

A METHOD FOR THE RELIEF OF PAIN IN TUMORS OF THE BRAIN.¹

BY WILLIAM BROWNING, M.D.,
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The hopeless character of most cases of this kind and the limited extent of our resources in treating them are generally recognized. A few cases can be approached surgically (with a view either to the radical removal of the growth, or occasionally to relief by drainage, or to the symptomatic relief of pressure that a trephine-opening affords). A small number respond somewhat to thyroid, a few remain latent, more are amenable to specific remedies, and many are temporarily benefited to an appreciable extent by iodide. The X-rays have been found in a few cases to temporarily numb the pain. These methods are more likely to prove palliative than radical. This brief summary about covers our scanty therapeutics, and even so makes a better showing than many may suppose possible. Granting all these exceptions, however, a majority of cases remains, and in these we are at a loss to know how to meet the conditions. In default of anything better, we grope around for means of symptomatic relief, and usually end by making continuous use of opiates in some form. The latter agents, however, are objectionable for several reasons; they obtund consciousness more or less, and in the long run aggravate pain by increasing constipation.

So long consequently as in the majority of cases of intracranial neoplasm we are unable to apply radical or curative measures, so long shall we need symptomatic agents. This is particularly true of the frequent and most distressing symptom, pain in or about the head. It is the sufferer's chief complaint, and it is especially for its relief that the present plan aims.

While the principal merit of the method to be suggested² is that of relieving symptoms, this is done in a less objectionable

¹Paper read before the Brooklyn Society for Neurology, Dec. 18, 1902.

²In the discussion of a paper at the 1901 meeting of the American Neurological Association, I briefly mentioned this means of relative relief in brain tumors. Attention had previously been called to the value of vaso-motor depressants and sedatives in the headache of syphilis (Brooklyn Med. Jour., 1889, 675), especially in the later forms, and that may have dated in part back to Seguin's use of aconitia and gelsemium for trigeminal neuralgia as early as 1879.

manner both from the patient's standpoint and from our own than is afforded by narcotics. Nor does it in any way interfere with the use of these if such becomes necessary.

One unquestionable advantage of the use, which, as you know, I have so strongly advocated for a number of years, of the vascular agents (whether to increase or depress the blood-pressure), is that they act in an intelligible way and not in some presumed or speculative manner. Their influence can be pretty definitely foretold and its effect controlled; hence the remedy can be fitted to the case. In other words, they are rational remedies and meet the demand for both honesty and exactness in practice.

The method in the present instance is very simple. It consists in the administration of such depressants as aconitia, veratrum³ or gelsemium⁴ in doses sufficient to soften and control the pulse. The amounts required are not far from those which were recommended in the abortive treatment of apoplexy (*N. Y. Medical Journal*, 1902, Feb. 15). The remedy can be continued as required, though the condition of the circulation should be frequently observed and the amount of the drug regulated according to the necessity of the case and the way it is borne. The dosage in any particular case is kept where it does not dangerously reduce the pulse on the one hand, and yet controls the pain on the other.

This plan may not at any and every instant completely stop pain, but it holds it within bearable limits, and may be all that is required even when the course of the trouble extends over many months. It appears to accomplish quite as much as the method of symptomatic trephining, which was proposed for a similar purpose, and to do this without any of the operative drawbacks.

Doubtless the thoroughness with which pain can be controlled in any given case will depend somewhat upon the manner in which it is produced. Clinically we have, besides those relatively fortunate cases without pain, certain types, and we can trace the source of the pain to at least three factors: (1) There is the local

³It has been suggested that, as veratrum has a tendency to produce nausea, it is undesirable for this purpose, unless it is the only agent of the kind at hand.

⁴My attention has been called to experimental work indicating that gelsemium "exerts no influence on blood-pressure." Clinically, however, it does affect the pulse, when given in full doses. A better effect might follow its combination with one of the others. As aconite can be given hypodermically it is as yet the best agent for our purpose.

and more or less general increase of intracranial pressure, as occurs most typically in intrasubstantial growths. Where the pain is thus directly due to pressure, it is less intense, but may be more continuous, like a deep ache. In this form marked relief may be expected. (2) Then there is the paroxysmal augmentation of arterial pressure in certain cases, to which reference will be made more fully. It is in these attacks doubtless that the terrific pain-seizures occur, and correspondingly this form should be relieved most of all by depressants. It is really here that we most need analgesics, and that opiates fail except to stupefy. (3) But when on the contrary the pain is the result of direct implication or stretching of external structures, as the meninges or outgoing nerves, and we get instead neuralgias and localized pains, then the good to be realized from depressants may be only proportionate to the degree in which pressure is a factor. And in fact it is supposed that in this last type of cases pain is especially likely to be felt.

It is fair to ask whether this plan has any injurious effect on the course of the trouble itself. Nothing of the sort has been apparent in any case, nor is there any theoretical reason for suspecting harm. So far as one can decide, life has if anything been prolonged. On paralysis if present it can hardly have much effect either way. Other, in part paroxysmal symptoms, as vomiting, dizziness, spasms, are as a whole favorably influenced. The growth of the neoplasm itself is not visibly affected, certainly not accelerated. Consciousness and the individual's personal control of himself is much better preserved. Moreover, remedies of this kind exert a very favorable influence on a certain tendency in these cases to brain-hemorrhage. This complication is specially frequent in and about the growth itself. Though such hemorrhages are more apt to be numerous than individually large, yet they decidedly aggravate the symptoms of the tumor, and anything tending to prevent their occurrence will be of advantage. Hence, aside from the pain, these remedies act on the other phases and manifestations of brain-tumor favorably if at all.

In occasional cases the depressive agents may also have some diagnostic or differential value.

The points that first suggested a trial of these agents were the increased intracranial pressure, and the pulsatory character

often of the pain. In such cases, where warranted by a strong pulse, it seemed proper to seek relief by depressants. Gradually it became evident that in most cases their use practically did away with the need for morphine. Recently a more striking basis for explaining the good effects in some conditions has been afforded by the work of Dr. Harvey Cushing⁵.

Perhaps his conclusions, so far as they bear on the present question, can be indicated by a few quotations. Speaking "of the alterations in the cerebral circulation as they are influenced by varying degrees of compression," he says: "When, however, the local process is in the near proximity of, or, if remote, when its effects are so far reaching that the vital centers of the bulb are compromised, the one symptom which with regularity is called forth, and which betokens a serious alteration in the local circulation, is a persisting rise in blood-pressure, which may or may not be associated with a pronounced vagus pulse, with respiratory changes, etc. The length of time experimentally during which these reactions can endure is considerable, even under the extreme degree of elevation—over 200 mm. of mercury from a level of 90 or 100 mm., in many cases—to which we have pushed the blood-pressure." This increased force of the current he figures as an effort of nature to overcome an obstruction. Yet the occurrence of this phenomenon presupposes an already existing increase of the intracranial pressure in the posterior fossæ—a condition that is frequently produced by tumors. The same paroxysmal or longer elevation of the blood-pressure may also have to do with the frequent occurrence of brain hemorrhages in these cases. And yet in the case of a brain tumor it does not appear that any particular good is accomplished by this heightening of pressure. Certainly it must augment any ache. It affords therefore an added indication for the use of depressants in easing and preventing the suffering of these patients.

It is hardly necessary to give details of cases; and the still inevitable ending makes their recital less inviting. If the argument appears sound, you can readily make use of the idea in suitable cases and arrive at your own conclusions. The claim that I present is simply that this is an improved palliative in a most distressing class of troubles.

⁵Amer. Journ. of the Med. Sciences, Sept., 1902.

TRICEPS, BICEPS, AND FINGER CLONUS.

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Theoretically, there is no reason why clonus of the triceps and biceps muscles should not be obtained when the tonicity of these muscles is increased, but such phenomena probably have not been described. The different text-books on nervous diseases (Gowers, Oppenheim, Mills, Strümpell), and Sternberg's¹ work on the tendon reflexes make no mention of the phenomena.

Triceps clonus was first obtained by me in the following case:

J. N., aged fifty-seven years, was admitted to the Philadelphia Hospital, April 1, 1903. At present he is in the insane department. Diagnosis: General paresis. History of syphilis and alcoholism was obtained. The symptoms began two and a half years ago. He has ideas of grandeur, hears voices, stumbles over syllables. He has some tremor of the lips, and Argyll-Robertson pupils, and the left pupil is larger than the right. The tendon reflexes are markedly exaggerated, and ankle and patellar clonus are obtained on both sides. He has no sensory changes, and no bladder nor rectal disturbances. Tapping the right triceps tendon above its insertion into the olecranon, produces a distinct clonus of that muscle, which is easily exhausted.

Triceps clonus was obtained more typically in a second case, and is fully described in the following report:

W. K., aged sixty-six years, was admitted to the service of Dr. William G. Spiller, in the Philadelphia Hospital, July 30, 1903. His father died with hemiplegia. The patient had not contracted syphilis, but was alcoholic. Four years ago he had a right-sided hemiplegia with aphasia. At present he drags the right leg when walking. The lower distribution of the right seventh nerve is paralyzed. The right upper limb is contrac-

¹Sternberg, M. Leipzig ü. Wien., 1893. "Die Sehnenreflexe und ihre Bedeutung."

tured at the elbow and wrist, and the fingers are flexed. The right knee is also contractured, but all these contractures can be overcome on forced passive movement. The tendon reflexes are very much exaggerated on both sides, more so on the right side. He has ankle clonus, but no patellar clonus. Babinski's sign is present. There is complete right-sided hemianesthesia for all forms of sensation. Incomplete hemianopsia is present. Atrophy is present in both sides, and is more marked on the paralyzed side.

The patient holds his paralyzed arm close to the chest, the forearm being flexed at a right angle to the arm, the hand being supported by the sound limb. On tapping the triceps tendon above its insertion into the olecranon a distinct clonus of the triceps muscle is produced, lasting at times three to four seconds. It is hard to count the vibrations with the eye or hand, but the number varies between twelve and twenty. Excitement increases the clonic movement, and at these times tapping even with the finger over the body of the muscle almost as high as its upper insertion, in fact a slight jar over any portion of the forearm or arm, will bring on the clonus. Indeed, the patient says that at times, while resting quietly, clonus of the triceps muscle will be produced independently of any stimulation.

The clonus consists in rapid extension and flexion of the forearm upon the arm, the triceps muscle stands out prominently, and in the rapid up and down movement of this muscle its tendinous insertions can easily be made out.

By striking the biceps tendon near the elbow joint with a percussion hammer, there is produced at times a clonus of the biceps muscle, causing flexion and extension of the forearm upon the arm. This is not nearly as constant as the triceps clonus, and can only be brought out on excitement. The resulting clonic vibrations are comparatively slow, there being from five to seven in two to four seconds.

Sometimes percussion over the biceps or triceps tendons will bring both clonus of the triceps and biceps muscles, the triceps clonus outlasting the latter. Again, striking the triceps tendon will at times bring on a clonus of the wrist besides the triceps clonus.

Clonus of the fingers is rarely found. Gowers² says that in an increased excitable state of the flexors of the fingers, a contraction, and sometimes by maintaining the increased tension, a clonus can be obtained. Sternberg merely mentions the possibility of finger clonus. No other mention of such a condition was found in the text-books referred to.

W. M., aged fifty-seven years, was admitted May 30, 1903, to the service of Dr. C. S. Potts, in the Philadelphia Hospital. He had right-sided hemiplegia, which developed twenty-six years previously. The man is chair-fast. The right upper limb is contracted at the elbow and wrist, and the thumb is turned inward, and the fingers are clenched. The right leg is contracted at the knee. All the tendon reflexes are increased. There are no sensory changes. Atrophy is more marked in the paralyzed side. Wrist clonus is not obtained on suddenly extending the tightly clenched fingers, the hand being held, but there is resulting clonic vibrations of the fingers upon the carpo-metacarpal joint. The movements are rapid, consisting in a flexion and extension, are easily exhausted, but soon return, the number of vibrations never being more than from five to seven, and lasting from one to one and a half seconds. Sudden extension of one finger or two fingers will not produce the clonus, but sudden extension of three or of all the fingers will.

I am indebted to my chief, Dr. William G. Spiller, for the privilege of reporting two of these cases, and to Dr. Potts for the privilege of reporting the third.

²Gowers, Sir W. R., 3d ed., p. 23, "Diseases of the Nervous System," vol. 1.

STUDIES UPON THE CEREBRAL CORTEX IN THE NORMAL HUMAN BRAIN AND IN DEMENTIA PARALYTICA.

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(Continued from page 648.)

Several neuroglia cells as at N, rounded or oval in shape, are to be seen here also interspersed among the nerve cells, and they increase in number proportionate to the nerve cell decrease as the white medullary center is reached. Several capillary blood vessels are to be seen in this layer, cut in different planes. Their walls are made up of a single layer of thin nucleated cells and the vessels filled with red blood corpuscles. The cells of this layer belong to the somatochrome class and either the archyochrome or gryochrome group, depending on whether one regards the chromatic substance as making up a distinct network as in the archyochrome group or as small granules arranged in threads or heaps as in the gryochrome group. It is difficult to determine a network of any distinctness in the majority of these cells, and they seem to the writer to belong more to the gryochrome group. The entire cortex thus measures in this section approximately 2.85 mm. in thickness as it is really an arbitrary division to state just where the gray cortex ends and the subcortical white medullary substance begins, there being a gradual transition from the one to other here as well as between the layers of the cortex. In order to obtain the approximate number and ratio of the nerve cells to the neuroglia cells an effort was made to count the same in various sections of the photomicrograph, counting the nerve cells first and marking each one as counted by black ink, and afterwards doing the same for the neuroglia cells. Upon subsequently using a Zeiss ocular micrometer divided into millimeter squares in a No. 3 ocular with a No. 6 objective, thus giving a magnification of 390 diameters, it was found that the former method, in which only a magnification of 100 diameters was used did not show all the structures present, and those shown were not sufficiently distinct at times to determine accurately their real nature, whether neuroglia cells, portions of nerve cells, etc. With the above magnification,—390 diameters,—and upon focussing in various planes, it was found for instance that a single mass upon the photomicrograph could be resolved into two or more neuroglia cells close to one another and in slightly different planes. By using the above combination it was found

that one side of the millimeter square in the eye-piece corresponded to .033 mm. of the stage micrometer and thus of the surface of the section under observation, so that a square millimeter of the ocular network was therefore equivalent to .03323, or .00999 square millimeters (practically .01 square millimeters, which latter decimal was used for convenience in this work) of surface of the section under examination. The ocular micrometer was divided into 36 squares and the number of nerve cells in each of these squares was counted and recorded in seven different fields, in this case in different parts of the section, thus making 252 squares in all. The result was added up and divided by 252 in order to give the average number of nerve cells for each square. This number was equivalent to that contained in .01 sq. mm. of actual surface of the section, or practically 1-100 of a square mm. The total number of neuroglia cells in 108 squares from three different fields was determined in the same manner, the resulting total being divided by 108 to give the average number of neuroglia cells for each sq. mm. of the ocular net-micrometer or .01 sq. mm. of actual surface of the section. These results multiplied by 100 thus will give the number of nerve and neuroglia cells for each square millimeter of actual surface of the section. With this method the average number of nerve cells to each sq. mm. of the ocular net-micrometer was found to be 1.1685; multiplied by 100 gives 116.85 as the average number of nerve cells to be found in each sq. mm. of this section in the second and third layers. In the region just below the middle of the second layer previously described as made up almost exclusively of small pyramidal cells these latter are found very densely packed together, in many parts as many as 2.7 cells were found to each square on an average. This result multiplied by 100 as before gives the high average of 270 nerve cells to each square millimeter in this narrow region, whereas the general average for the entire second and third layer, not including this region, is but 93 nerve cells to the sq. mm., thus showing the great variation in regions of the same section closely approximating one another. The nerve cells of the first layer were so few and scattered that they were not included in this count. In the same way the average number of neuroglia cells to the square millimeter of surface of the section was found to be 125. Counts were also made from a section from the first frontal convolution, somewhat anterior to that from which Plate II, Fig. 3, was taken. This latter block was hardened in alcohol, the section was 10 microns in thickness, and similarly stained with methylene blue. Decolorization was not carried on to such an extent, so that the neuroglia cells were not decolorized, and the section being 10 microns in thick-

ness in comparison to 6-2-3 microns in Plate II, Fig. 3, the neuroglia cells were found to be more numerous. This would seem to indicate that fixing in Van Gehuchten's fluid and staining the same length of time in warm methylene blue results in a somewhat greater decolorization of the neuroglia cells in the following differentiation than in sections hardened in alcohol and treated similarly in respect to staining and differentiation. As stated this section was 10 microns in thickness, whereas that of Plate II, Fig. 3 is but 6-2-3 microns in thickness; but it will be seen by the following figures that whereas there is only a slightly greater number of nerve cells in the former as compared to the latter the neuroglia cells are considerably in excess in the former. Here counts were made in seven different fields of 36 squares each in various parts of the second and third layers for the nerve cells, and in all three layers for the neuroglia cells. The average number of nerve cells was found to be 1.27 to each sq. mm. of the ocular net-micrometer, which multiplied by 100 gives 127 as the average number of nerve cells to the sq. mm. of surface of the section. In the same way the average number of neuroglia cells was found to be 1.64 to the square millimeter of the ocular net-micrometer, which again multiplied by 100 gives 164, as the average number of neuroglia cells to each sq. mm. of surface of the section. The difference in the number of nerve cells in the two sections is not very great,—about 10 per cent, but in the number of neuroglia cells it is much more marked,—over 30 per cent, due partly to the difference in thickness and partly to the greater decolorization of the first section described (Table II). Hammarberg (1895) for counting nerve cells, and Popoff (1894) for determining the ratio of increase in neuroglia cells in acute and chronic cases of Asiatic cholera, used the ocular net-micrometer in a manner similar to that made use of in this article, but in each case in a different line of investigation; the former in the study of idiocy, and the latter, as already stated, in Asiatic cholera. The number of nerve cells and of neuroglia cells, as well as their ratio to one another, varies in the different layers of the cortex. In the first layer there are but few nerve cells, whereas the neuroglia cells are about the same or even more in number than in the other layers, thus making a high ratio. In the second and third layers, however, the cells are the important elements and occupy the greatest amount of space, and although fewer numerically than the neuroglia cells the latter are much smaller, less conspicuous, and the ratio is not so great. Then again the cells are not evenly distributed, but have a tendency to be arranged more or less in irregular groups, so that in some places they are relatively widely scattered, whereas in others they are more closely ag-

gregated, so that a number of counts in various parts of the field are necessary to get an approximate average of the number of cells. The thickness of the section, degree of staining, and extent of differentiation or decolorization must also be considered in Nissl work, as too great decolorization will prevent some cells from being recorded in a photomicrograph, and an extremely thin section would not contain as many cells as a thicker one. Sections 6-2-3 and 10 microns in thickness (two

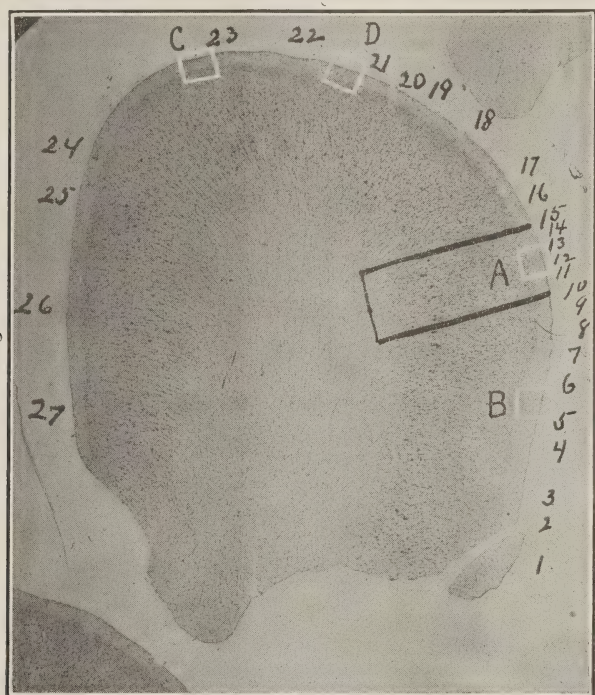


Plate III, Fig. 7.

and three clicks of the Minot microtome) were used in this work, and when well stained and decolorization is not carried too far, will show all cells to be found in such a plane.

Central Region Turning now to the region posterior to this we come to the motor region of the cortex, comprising the anterior and posterior central convolutions. This region is of especial interest in many ways, and has probably been more extensively studied than any other of the various regions of the cortex. Plate III, Fig. 7, is a photomicrograph magnified 14

diameters, taken from a section 10 microns in thickness at the upper third of the anterior central convolution of Brain A. (Plate I, Fig. 1, 2.) This block was also fixed in Van Gehuchten's fluid, imbedded in paraffin and sectioned serially by a Minot microtome. This section was then stained with warm methylene violet, differentiated in alcohols, 30, 50, 70 and 95 per cent., and absolute, cleared in xylol and mounted in xylol-damar. The actual size of the section which includes the entire width of the gyrus, is .7 of a c. m. in width by .7 c. m. in depth, and taken from a block .4 cm. in thickness, the plane of the section being at right angles to the direction of the gyrus. At the point *a* and outlined in ink is the strip seen in Plate III, Fig. 8, at a magnification of 100 diameters. The outer layer of the cortex is seen to be fairly uniform in thickness, with the exception of the regions indicated at *b* and *c*. At *b* this layer is considerably thicker than in any other part, gradually diminishing in thickness near *a* where it is of the average thickness. At the point *c* is seen a slight thinning of the cortex, which in Plate IV, Fig. 9, results in the entire disappearance of this layer and is intermediate in degree in Plate IV, Fig. 11. This condition will be more fully discussed in the description of these latter plates, and it will be simply stated now that this is an artefact probably due to manipulation. The general radiating appearance of the cortex from the white medullary center to the periphery is well seen here. In the region of the lower part of the second or middle cortical layer even with this low magnification can be distinctly seen the giant pyramidal, or Betz cells arranged singly or in groups. Below this the cortex becomes paler and finally merges into the central medullated white substance. This magnification is especially good for studying the arrangement of the Betz cells. Beginning with the first group in the lowest part of the section on the right, which is the posterior surface of the gyrus, opposite 1 is a single Betz cell of moderate size; just above this, opposite 2 is a group of two Betz cells, one nearer the surface and above the other in the lower part of the second or pyramidal cell layer. Somewhat higher up, opposite 3, and somewhat deeper, are seen two very large Betz cells, one deeper and further from the surface, as well as higher up than the other. Opposite 4 are three Betz cells too far separated to be considered as a single group, so must be regarded as solitary cells. Just above them at 5 are three smaller cells, one above the other at about the same depth of the cortex. Opposite 6 are two small Betz cells, one nearer the surface than the other. Then for a considerable distance no Betz cells are to be seen until we reach a point opposite 7 where a fair sized solitary cell is made out. Just above this

opposite 8 are two rather small Betz cells, and a short distance above at 9 a group of three containing two large and one small Betz cell. Opposite 10, and just below the lower ink mark are two very large Betz cells, one overlapping the other, giving the appearance of one in the plate. Just above the ink line opposite 11 is to be seen a group of four Betz cells, one large, and the other three smaller. These and those above and included between the two parallel ink lines can be seen at a magnification of 100 diameters in Plate III, Fig. 8, this group being on the extreme right of the latter. Opposite 12 is a group of four Betz cells arranged radially, the upper of the two in the middle being the largest, and the lowest next in size. Opposite 13 is a large solitary Betz cell, and above this and opposite 14 is a group of three Betz cells. Opposite 15 is another large solitary Betz cell, below which, and a little deeper in the cortex between it and the previously described group, is another Betz cell of medium size. Just above this is a group of two smaller Betz cells. Opposite 16 are three large cells in a group which are just to the left of Plate III, Fig. 8, and are intermediate in type between the well defined Betz cells and the ordinary large pyramidal cells, above this and opposite 17 is another group of four cells similar in type. Opposite 18 is a group of two Betz cells, one considerably larger than the other and nearer the surface of the cortex; in the same radial line is a medium sized solitary Betz cell. Just above this are five Betz cells opposite 19 and radially arranged in a row. Opposite 20 is a solitary Betz cell, and just above opposite 21 is a group of two Betz cells, one very large and the other quite small. Above this (to the left) are a number of pyramidal cells scarcely large enough to be considered as Betz cells, although as previously noted there are intermediate forms which might be considered as very small Betz cells or very large pyramidal cells. Opposite 22 are seen three Betz cells rather scattered, the one in the middle being at a deeper level of the cortex. Opposite 23 is seen a small solitary Betz cell, and opposite 24 is another solitary Betz cell which is on the anterior aspect of the gyrus. Between it and the last described cell are quite a number of large cells intermediate in type between the Betz cell and the largest pyramidal cells. Opposite 25 is still another solitary Betz cell. Then comes a region for a considerable distance in which are seen several large cells at this level, but hardly large enough to be considered as Betz cells until we reach that part of the cortex on the anterior aspect of the convolution opposite 26, where we see a group of two made up of a large Betz cell and a smaller one. Finally below this, at a considerable distance and opposite 27, is a group of three Betz cells, the one in the middle being large,

and the other two small. It will be seen that the Betz cells are larger and more numerous on the posterior aspect of the gyrus than upon the anterior aspect. There are thirty-one groups of these cells in all, 13 of which contain but a solitary cell, so can hardly be considered as a group, though of course many of them form part of a group, the remaining cells being in another plane. Ten of these groups contain two cells each, 5 cells each, and but two groups have four cells each, and one group has five cells, making 61 Betz cells in all in this section. Beginning posteriorly and following around to the lowest group on the anterior surface, the number of cells in each group as seen in the same plane is as follows:—1, 2, 2, 1, 1, 1, 3, 2, 1, 2, 3, 2, 4, 4, 1, 3, 1, 1, 2, 2, 2, 1, 5, 1, 2, 3, 1, 1, 1, 2 and 3. These cells lie deeper in the cortex and thus at a greater distance from the surface in the posterior aspect of the gyrus than at the vertex or at the anterior aspect. Of course the number of cells in this one plane does not represent in every case all the cells in a group, but only those in this particular plane. As many of these groups are more or less spherical no one plane will show more than a part of such a group, and as stated above some of these solitary cells are the outer members of some of these groups.

Plate III, Fig. 8, as previously stated, shows the strip *a* of Plate III, Fig. 7, under a magnification of 100 diameters and in strong contrast to Plate II, Fig. 3, is to be seen the much larger number of neuroglia cells, the methylene violet stain seeming to have a stronger affinity for the neuroglia cells than the methylene blue stain. The cortex in this plate is also seen to be more condensed and not so deep as in Plate II, Fig. 3. The first layer measure but .20 mm. in thickness in contrast to .25 mm. in the above mentioned plate. In this layer neuroglia cells are more numerous, with a few scarcely scattered nerve cells only to be made out under high magnification, and similar to those found in the first layer in Plate II, Fig. 3. The second or pyramidal cell layer is of special importance and interest. It is deeper than the corresponding layer in Plate II, Fig. 3, measuring 1.75 mm. in thickness as contrasted to 1.40 mm. in the former. Here too in addition to the large and small pyramidal cells are found the giant pyramidal or Betz cells, irregularly distributed, singly or in groups, and at different levels, so that it is impossible to describe them as forming a distinct layer, although in this plate, with the exception of one cell, at *a* they appear to be placed in the lower part of this portion of the cortex now being described. In taking one section after another, however, it will be found that these cells are located at different levels, the majority being in the lower portion of this second layer. They furthermore have a tendency to be arranged in groups containing from two

to ten cells, rarely more, and finally we find some sections containing no Betz cells at all. The pyramidal cells in the motor area are found to be larger in size on an average than those of the frontal region. The chromatic substance is not arranged in such large distinct bodies as in the Betz cells, but may be seen distributed in clumps of finer or coarser granules in different parts of the cell-body, especially about the nucleus and about the base, and at the beginning of the dendritic processes in the cell-body. Fig. M (M, of Plate III, Fig. 8), shows one of these cells under a high power, X1300 (No. 3, ocular, 1-12 inch oil immersion objective, Leitz). Here the finely granular chromatic substance is distributed in a ring about the nucleus, a clump is seen at the base to the left where a dendritic process is given off and also clumps are seen at the base of the dendritic process on the right and other clumps in the upper part of the cell-body and in the apical process. In addition to this, small sized linear and rounded chromophilic bodies are seen in the upper part of the cell-body and apical process and also in the dendritic processes given off at the base. The nucleus here is large, rounded, centrally placed, and contains a round, deeply stained, prominent nucleolus, surrounded by slightly stained homogeneous nuclear substance. In some cells the finely granular chromatic substance predominates in the cell-body, while in others the small chromophilic bodies are more prominent, whereas the majority contain both, as in the above described cell. In some of the smaller pyramidal cells the large nucleus almost fills up the cell-body, with but a narrow rim of finely granular chromatic substance surrounding the same and extending into the apical and basal processes. These latter cells would be classified under the group of karyochrome nerve cells; whereas the larger pyramidal cells with the stainable substance arranged in striæ in the same direction as the contour of the cell-body come under the head of the stichochrome nerve cells of the somatochrome class. Turning to the Betz cells and examining these under a high power (No. 3 ocular 1-12 inch oil immersion objective, Leitz. X1300) they are seen to contain numerous distinct chromophilic bodies varying in size and shape. In Fig. N (N, of Plate III, Fig. 8) the upper part of the cell is not included in this section. From below are seen two dendritic processes given off from the under side of the cell-body, that is having the cell-body from a lower plane than figured in the drawing, upon the right side; and one slightly larger process from the left side. These at intervals contain distinct chromophilic granules, for the most part linear in shape, with the long axis parallel to the axis of the process and extending for a considerable distance into the process—as far as they could be

traced in the section. About the middle of the left dendritic process is seen a large irregular chromophilic body at a point where a branch is probably given off, as wedge-shaped chromatic masses are often found at the point of branching of a dendritic process, Nissl's so-called "wedges of division." Within the cell-body the granules are seen to be elongated, rounded or irregular in shape, the first mentioned predominating; some much larger than others, and with the general arrangement of the long axis parallel to the sides of the cell-body. The nucleus is large, well defined, and placed in the upper part of the cell-body and nearer the right than the left side. There is a well defined, deeply stained nucleolus centrally placed and surrounded by a homogeneous, slightly colored nuclear substance, with no well defined network. At the base of the cell-body, occupying a space somewhat larger than the cell nucleus is a deposit of yellowish pigment, elliptical in outline, and entirely displacing all other cell contents. No other pigment is to be found in this cell. The arrangement of the chromophilic granules in striæ in the same direction as the contour of the cell-body classifies it as a stichochrome nerve cell of the somatochrome class. Fig. o (o of Plate III, Fig. 8) shows another of these Betz cells examined under the same magnification of 1,300 diameters (No. 3 ocular, 1-12 in oil immersion lens, Leitz), in which the arrangement of the chromophilic bodies in striæ parallel to the contour of the cell-body is very well marked. This arrangement extends far out in the basal process and high up in the apical process. The nucleus is situated more on one side of the cell-body than the other, and the nucleolus is not included in this plane. There are three dendritic processes to be seen, the apical extending for a considerable distance towards the periphery, and gradually decreasing in size, and two basal processes, the one to the left unbranched, while the one on the right gives off a large branch shortly after leaving the cell-body, at which point *a* is one of the so-called "wedges of division" of Nissl. There is no pigment in this cell, but in the one seen just below this in Plate III, Fig. 8, marked P, a large mass of yellow pigment is seen above and to one side of the nucleus at the base of the apical process. There are but few chromophilic bodies in this cell compared to the two previously described, the chromatin being for the most part in the form of fine granules arranged in larger and smaller masses in different parts of the cell. The Betz cell seen lying just below and to the right of this latter, and marked Q in Plate III, Fig. 8, also contains a small mass of pigment poorly defined, and lying just below the nucleus at the base of the cell. The other Betz cells seen in this plate contain no pigment. While discussing the Betz cells, and before proceeding to the description of the third layer, several other photomicrographs will be described as illustrating the

grouping and structure of these cells. The first of these, Plate IV, Fig. 9, is a photomicrograph magnified 14 diameters of a section in close proximity to that seen in Plate III, Fig. 7, of the same size as this latter and treated in the same way, excepting that methylene blue was used as the stain here. Opposite *a*, and enclosed in ink lines, is the segment of the section shown in Plate IV, Fig. 10, under a magnification of 100 diameters. The other layer is seen to vary in thickness, being much thicker at the posterior aspect, at *b*, than at the vertex or the anterior aspect.

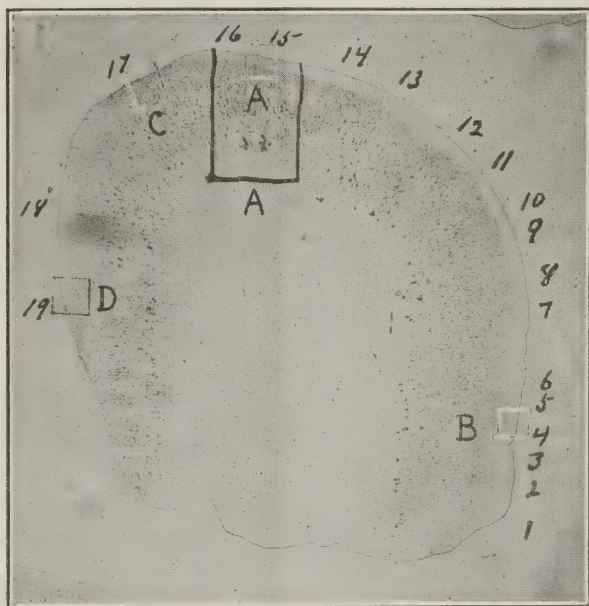


Fig. J.

At *c* and *d* the second layer is seen at the surface, due no doubt to injury in manipulation, probably more from pressure than actual tearing, as the surface is smooth and continuous at both ends, with the adjacent surfaces. No doubt artefacts similar to this, and of greater or less extent, must be carefully guarded against and considered in all work upon such a delicate and yielding tissue as the brain, especially when secured within a few minutes after death, as in this case, and before any post-mortem fixity results. The second layer shows the radial arrangement of the cells, but the most conspicuous feature is the distribution and arrangement of the Betz cells contained in the lower part

of this layer singly or in groups of varying number and size. Below this the less conspicuous third layer is gradually lost to view as it merges into the white medullary substance. The arrangement of the Betz cells will be described in detail as photomicrographs of this magnification, 14 diameters, show their distribution especially well. Beginning at the posterior portion of the convolution below the letter *b*, the first large Betz cell is to be seen opposite the figure 1, and is solitary, whereas just above and opposite 2, is a group of four Betz cells all of about the same depth of the cortex. Above this, opposite 3, is a large solitary Betz cell. Opposite 4 is a group of three cells arranged radially above one another. Opposite 5 is another solitary Betz cell. Above it a small portion of a Betz cell is seen which lies mostly in another plane, while below it small portions of three Betz cells are seen lying for the most part in another plane. Above this, and opposite 6, is a group of two Betz cells at the same depth, and one just above the other. Between 6 and 7 are seen cells, two in number, which might be regarded as intermediate in character between the large pyramidal and Betz cells, having a similar arrangement of chromophilic substance, but of intermediate size. Opposite 7 is a group of two Betz cells, and opposite 8 is another group of two Betz cells somewhat nearer the surface, and just under this latter group and deeper in the cortex is a solitary Betz cell. Opposite 9 is a group of two Betz cells, only a very small portion of the upper cell being seen in this plane. Opposite 10, a portion of a solitary Betz cell is seen, and opposite 11 is still another solitary Betz cell. Opposite 12 is a group of three Betz cells, and just to the right of this and at the same level, is a part of a solitary Betz cell, while nearer to the surface and in the same radial line is still another small solitary Betz cell. Opposite 13 is a group of six Betz cells, two of which are large, and appear very prominently, the other four lying somewhat to the right, being smaller, but having the characteristic structure of the Betz cells. Opposite 14 is a solitary Betz cell, and next we come to the two groups seen under a magnification of 100 diameters in Plate IV, Fig. 10, and in this plate seen enclosed in ink lines and opposite figures 15 and 16. The first group, or that opposite 15, contains nine well defined Betz cells, while the adjacent group, opposite 16, contains six Betz cells, three large cells and the segments of three others, the greater part of these latter lying in another plane. Opposite 17 are four scattered cells at about the same depth of the cortex, and intermediate in size and structure, between the large pyramidal and the Betz cells. Opposite 18 is a large solitary Betz cell, above and below it being the cells intermediate in type just described. At 19 is a group of six, made up of one large Betz cell and five smaller ones about it. Above and below this are other pyramidal

cells at this level, and of the above described intermediate type. There are in all fifty-nine of these Betz cells in this section, arranged singly or in groups; beginning posteriorly at 1, and proceeding around to the anterior and last group at 19, as follows: 1, 4, 1, 3, 1, 1, 3, 2, 2, 2, 1, 2, 1, 1, 3, 1, 1, 6, 1, 9, 6, 1 and 6, thus making twenty-three groups in all, eleven of which contain solitary cells, four contain two cells, three

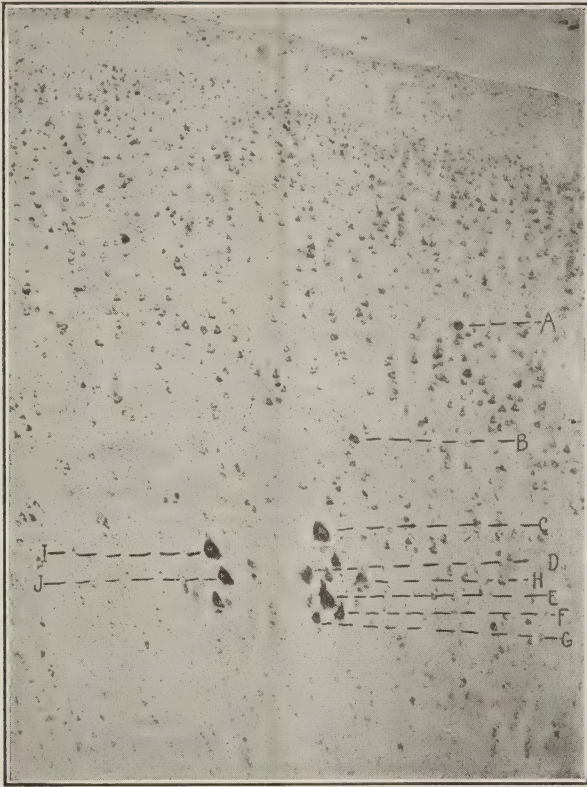


Plate IV, Fig. 10

contain three cells, one contains four cells, three contain six cells, and one nine cells, in the plane of this section. Of these cells thirty contain distinct pigmentary deposits, these being practically all of the larger cells. Quite a number of the others in the plane of the main portion of the cell would undoubtedly also contain pigmentary deposits. The grouping, of course, is here described for only the plane of this section and the probability is that some at

least of these solitary cells are a part of a group,, the remaining cells lying in another plane. Furthermore, some of the groups described may contain additional cells lying in the plane above and below that of this section. Plate IV. Fig. 10, previously mentioned, has a magnification of 100 diameters, and includes the strip *a* outlined in ink in Plate IV. Fig. 9. Two groups of Betz cells are seen, one containing nine and the other six cells. Two other cells, *a* and *b*, though smaller yet in structure approach in charac-

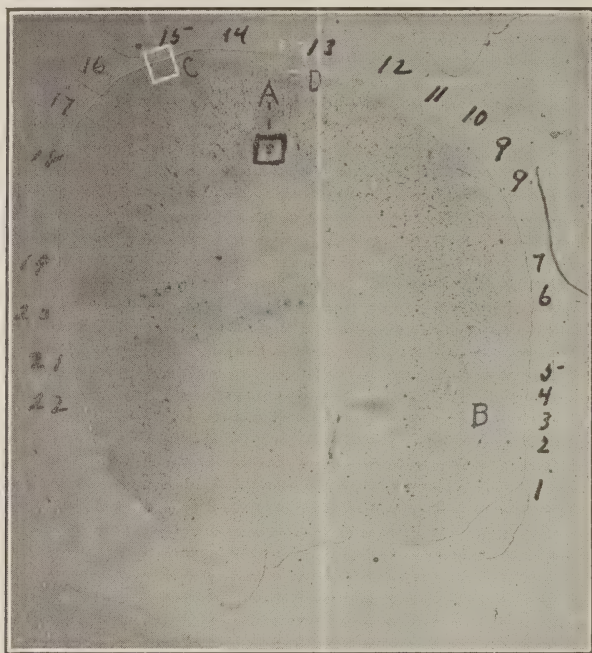


Plate IV, Fig. 11

ter the Betz cells. It will be noticed that the cortex is not so deep in this plate, the first layer being but .175 mm. in thickness, and the second but 1.20 mm. in thickness in comparison to .20 and 1.75 mm., respectively, in Plate III. Fig. 8. It will be noted, however, that this strip was taken from the vertex of the section where the cortex is not so deep as on the posterior aspect from which Plate III. Fig. 8 was taken. These two groups of Betz cells are but 1.20 mm. from the surface of the cortex here in contrast to 1.70 mm. in Plate III. Fig. 8, showing the variability in thickness of the cortex in close proximity, as both plates were made from

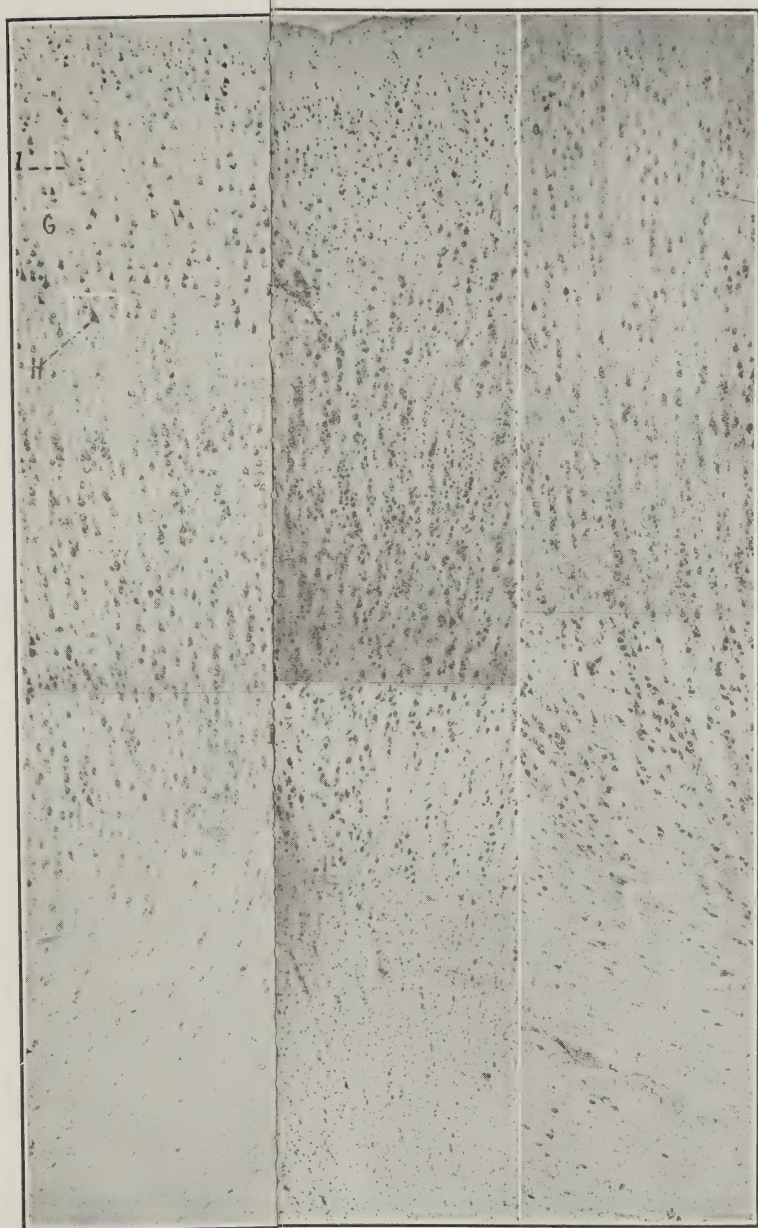


Plate II, Fig. 3. Plate VII, Fig. 21.

Plate VIII, Fig. 23.

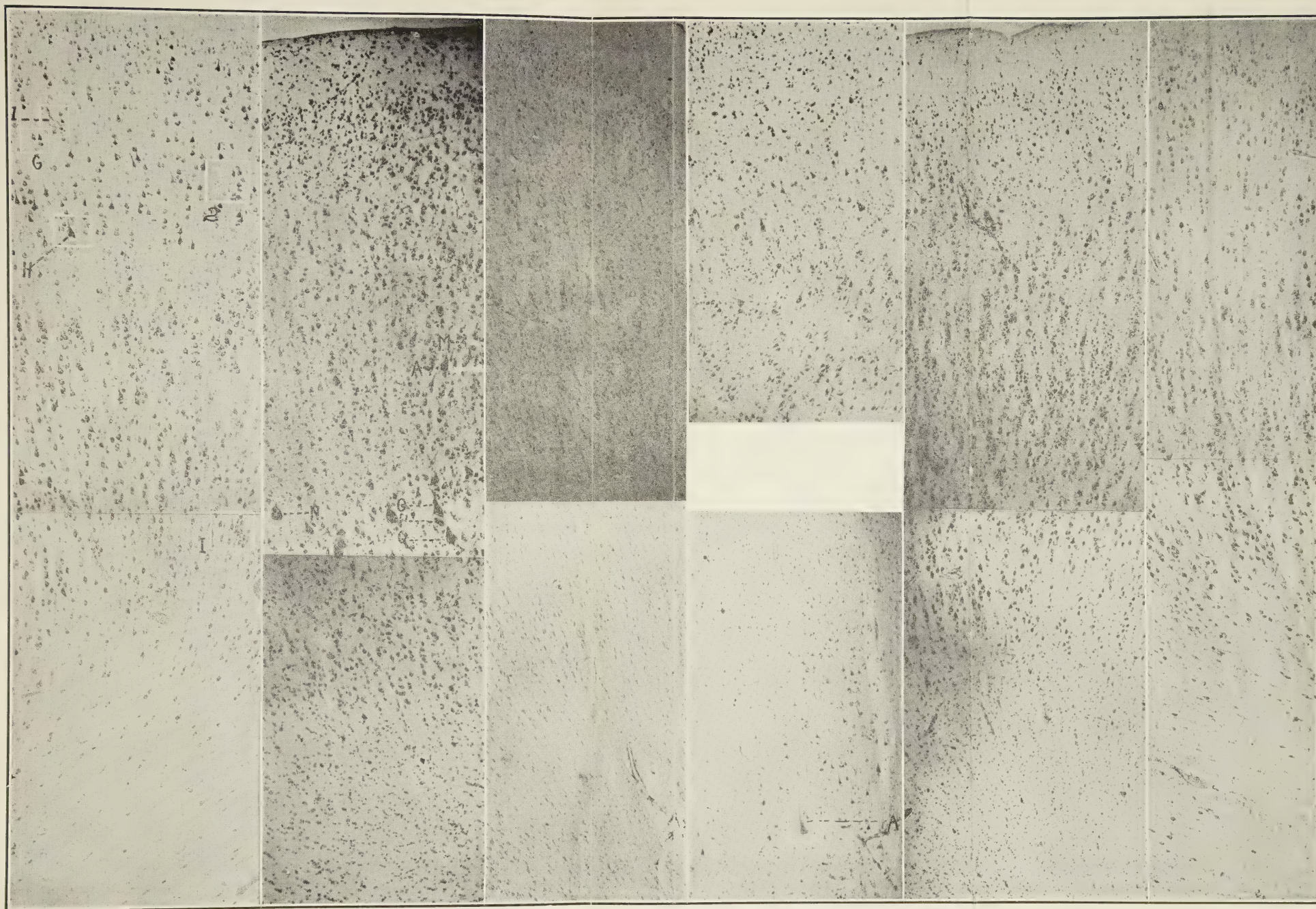


Plate II, Fig. 3.

Plate III, Fig. 8.

Plate VI, Fig. 17.

Plate VII, Fig. 19.
Plate XIII, Fig. 41.

Plate VII, Fig. 21.

Plate VIII, Fig. 23.

sections cut from the same block which was not more than half a centimeter in thickness. The Betz cells here have the same structural characteristics as those described in Plate III. Fig. 8, and five of the cells, marked *c*, *d*, *e*, *f*, and *g*, of the larger group, and two of the cells, marked *i* and *j*, of the smaller group, contain distinct yellowish pigment. The larger of the two groups and containing nine Betz cells is made up of two very large cells cut in the plane of the nucleus, four smaller cells also cut in the plane of the nucleus, while the remaining three cells show only a small portion of the body of each cell here, the large portion of these latter being in another plane. Five of these nine cells contain deposits of pigment, and all show the chromophilic granules, being seen especially well in the cells marked *c*, *d*, *e*, and *h*. The cells in this group are quite close to one another and do not take up a very large space. The smaller group to the left contains six Betz cells, three large and conspicuous, and three others, one to the right and two to the left of these large cells which lie only partially in this plane and are small in size. Two of the three large cells marked *i* and *j* contain deposits of yellowish pigment at the base of the body of the cell, and all contain well defined chromophilic bodies. Plate IV. Fig. 11 is another photomicrograph of a section from the same block as that from which the sections represented in Plate III. Fig. 7, and Plate IV. Fig. 9, were taken. This section is 10 microns in thickness, stained with methylene blue and otherwise similarly treated as the above mentioned plates. This magnification of 14 diameters shows the outer layer to be fairly uniform in thickness, excepting at the points *b* and *c*. At *b* it is of relatively the same thickness as at the same point *b* in Plate IV. Fig. 9, about one and one-half times as thick as at *d* into which it gradually merges. At *c* where no outer layer is to be seen in Plate IV. Fig. 9, here the outer layer is distinctly seen but somewhat thinner than on either side, showing the artefact to be less in degree in this section. At *a* the group of cells represented in Plate V. Fig. 13, is seen, and each cell can easily be distinguished and counted under this magnification of 14 diameters. The second layer here is also especially interesting in respect to the size, number and distribution of the Betz cells singly or in groups in the lower portion of the layer near the transition into the third or spindle cell layer. This latter is less distinct and lost below in the medullary white substance in the interior of the gyrus. A detailed description of the arrangement of these Betz cells will be given in order to compare the same with those of Plate III. Fig. 7, and Plate IV. Fig. 9. Beginning with the first group at the lowest part of the section on the right, which is the posterior surface of the gyrus, opposite 1, is seen a group of three large Betz cells. Above this some distance opposite 2, is a solitary Betz cell. Opposite 3, is another group of three Betz cells at a slightly greater

depth. Above this and opposite 4 and 5, respectively, are large solitary Betz cells. Then there is a considerable interval where there are a number of large pyramidal cells but scarcely approaching the size of a Betz cell until the point opposite 6 is reached where a large solitary Betz cell is seen and just above it a group of two Betz cells somewhat smaller in size is to be noted. Opposite 7 is a solitary Betz cell at a greater depth. Just above this and slightly nearer the surface are several intermediate type cells arranged in two rows, one at a slightly greater depth than the other. Opposite 8 and at the same level as the preceding is a solitary Betz cell, and above this opposite 9 is a larger solitary Betz cell. At the same depth and slightly nearer the vertex is a group of two Betz cells, one large and the other small. About midway between these and the surface of the cortex are seen several very large pyramidal cells, the so-called intermediate type between the Betz cells or giant pyramidal cells and the ordinary sized large pyramidal cells. Opposite 10 is seen a group of two very large Betz cells just below and to one side of a large blood vessel. Just above this and nearer the surface is a small solitary Betz cell. Opposite 11 are three groups of Betz cells, the first containing three, the second two, and the third above and nearer the surface consisting of a large solitary Betz cell. Opposite 12 is a large solitary Betz cell, considerably nearer the surface than the groups just described. A group of two large Betz cells, one just above the other, is seen opposite 13. This brings us to the large group opposite *a* and seen under a magnification of 850 diameters in Plate V. Fig. 13, containing eight Betz cells which will be described in detail later when discussing this latter plate. To the left of this group opposite 14 are seen three Betz cells of small size and somewhat separated from one another. To the left of these at a short distance are seen two Betz cells, one above the other. Opposite 15 are seen two groups of two each, the cells in the first group being somewhat separated from one another and one above the other. Opposite 16 is a well marked group of four Betz cells, two being very large and the other two smaller in size. Below this and upon the anterior surface of the gyrus opposite 17 are two solitary Betz cells, one lower than the other and nearer the surface. Opposite 18 is still another smaller solitary Betz cell at a greater depth. For some distance below this no Betz cells are to be seen until opposite 19 is a solitary Betz cell. Just below this and slightly nearer the surface is a group of three of the intermediate sized cells. Opposite 20 are two large solitary Betz cells, the lower larger one being at a slightly greater depth. Then comes another region barren of these cells until the point opposite 21 is reached where a small solitary intermediate size cell is seen, and below this a group of two of these same cells is to be noted, one at a greater depth than

the other. Opposite 22 is a very large solitary Betz cell. Just below this are two intermediate size cells, and still below this and at a slightly greater depth are three of these intermediate size cells. This includes all the Betz cells though, as above noted, here and there in different parts of this layer; but for the most part in the same relative position, are to be seen very large pyramidal cells approximating these Betz cells both in size and structure, and the writer thinks should be regarded as intermediate in type between the large pyramidal cells and the giant pyramidal or Betz cells,—a transition form, if they might be so called. There are twenty-eight groups of these Betz cells in all in this plate, varying in number from one to eight cells in each group, one to three being the usual number. Fourteen of these groups contain but one cell each, and these so-called solitary cells are usually of large size. There are eight groups containing two cells each, four groups of three cells each, but one group containing four cells, and one group containing eight cells. Taking the groups from the lower portion of the posterior surface in succession, as above described, and the number of cells in each of the twenty-nine groups is found to be as follows: 3, 1, 3, 1, 1, 1, 2, 1, 1, 1, 2, 2, 1, 3, 2, 1, 1, 2, 8, 3, 2, 2, 2, 4, 1, 1, 1, 1, 1, making a total of fifty-five Betz cells in the section, and thus averaging about two cells to each group. The number of groups is somewhat more than in Plate IV. Fig. 9, in which there were twenty-three groups containing in all fifty-nine Betz cells, and somewhat less than in Plate III. Fig. 7, where there are thirty-one groups containing sixty-one cells. It will thus be seen that the average number of groups and total number of cells in these three sections from near-by portions of the same block are fairly uniform in regard to the arrangement and distribution of the Betz cells. It is to be noted that more cells are found on the posterior aspect of the gyrus than on the anterior. In Plate III. Fig. 7, there are fifty cells arranged in twenty-five groups on the posterior aspect of the gyrus, with but four cells in two groups at the vertex, and seven cells in four groups on the anterior aspect. In Plate IV. Fig. 9 there are thirty-seven cells arranged in nineteen groups on the posterior aspect of the gyrus, fifteen cells in two groups at the vertex, and seven cells in two groups on the anterior aspect. Finally, in Plate IV. Fig. 11, there are twenty-seven cells arranged in seventeen groups in the posterior aspect of the gyrus, nineteen cells in six groups at the vertex, and nine cells arranged in six groups on the anterior aspect. It will thus be seen that most of the Betz cells are on the posterior aspect of the gyrus and gradually diminish in number upon approaching the vertex until upon the anterior aspect there are but few to be seen. Table III. shows the above results in tabulated form. Upon referring to this latter it will be seen that the average number of cells in each section is fairly uniform—55, 59

and 61 for the three sections; the grouping is somewhat more variable—23, 29 and 31, most of the cells singly or in groups of two or more being upon the posterior aspect and vertex. This corresponds to the findings of Lewis and Clarke ('78) who examined sections from various parts of the left anterior central convolution and the posterior portion of the two upper frontal con-

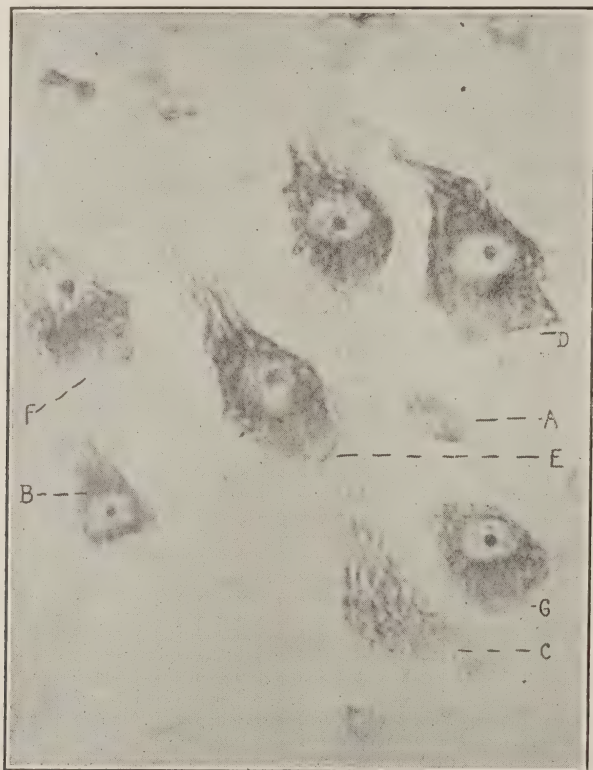


Plate V, Fig. 13

volutions on the same side in several human brains microscopically, and made diagrams of the distribution of the Betz cells at various points instead of employing photomicrographs showing the number, arrangement and relation to other elements of the cortex of these Betz cells as in this article. Plate V. Fig. 13 is a beautiful photomicrograph of a group of these Betz cells magnified 825 diameters and taken from the point marked *a* in Plate IV. Fig. 11. Here is seen a "nest," or group of these cells, eight

in number. All of these Betz cells contain pigment excepting the one marked *a* which in size is not much larger than one of the large pyramidal cells, but in structure resembles the Betz cells. This pigment, varying in amount, is situated at the base of the cell below the nucleus, excepting in the cell marked *b* where it is just above the nucleus. In the cell marked *c* it extends down into the large basal dendritic process, given off to the right for a short distance. In the cells marked *c*, *d*, *e*, *f* and *g*, the cell body appears of a lighter color in the photomicrograph where this yellowish pig-

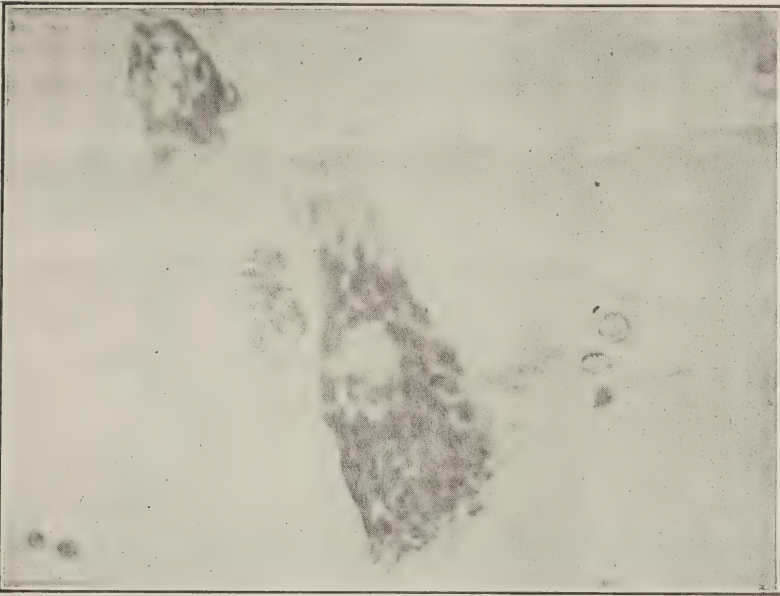


Plate IV, Fig. 12

mentary deposit is found. The cell marked *c*, sectioned outside of the plane of the nucleus, shows very well the general arrangement of the rounded or elongated chromophilic bodies in a direction parallel to the contour of the cell-body, thus classifying it with the others of similar type as stichochrome nerve cells of the somatochrome class. The cell marked *e* shows the general parallel arrangement of the chromophilic bodies in the apical process very well. The group, as a whole, is very compact and as each cell is cut in a somewhat different plane from the others most of the features of such a "nest" are thus brought out. Of course, there may be other cells in this group lying outside of the ones seen here—in the planes above and below this. Plate IV. Fig. 12 is a pho-

tomicrograph of a Betz cell magnified 1,400 diameters from a section adjacent to and upon the same slide as that from which Plate III. Fig. 8 was taken. The section is, therefore, similar in shape to the one in Plate III. Fig. 7, and this cell is situated in a position opposite to the letter *d*, and in the lower portion of the second

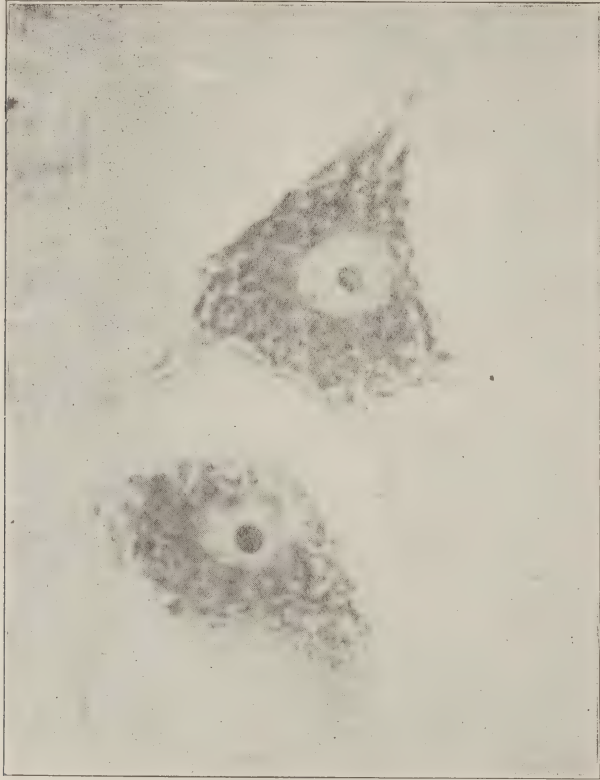


Plate V, Fig. 14

or pyramidal cell layer of this latter. The section is ten microns in thickness and stained with methylene violet. It can readily be appreciated from this photomicrograph that only an extremely thin plane can be seen under such a high magnification and not all of this even will be in sharp focus. The nucleus here is mostly below the plane of the section and the deeply stained nucleolus entirely so. Two dendritic processes are faintly seen coming off from the base of the cell, the one on the right but faintly shown as it is at a different level for the most part. The one on the left

is somewhat more distinct and chromophilic bodies are found to enter the base and extend for a considerable distance into the same. These bodies of varying size and in general rounded or linear in shape, are closely packed in most parts of the cell-body, excepting at the base where a large deposit of yellowish pigment, taking up about the same area of space, as the nucleus, is to be found. These chromophilic bodies are arranged in general parallel to the contour of the cell-body and extend into both the basal dendritic processes and the apical process as far as they can be traced. A portion of another Betz cell smaller in size is seen above and to the left. About the larger cell are several neuroglia cells singly or in groups, and these are seen to be distinctly rounded or oval with usually a ring of chromatic substance of varying thickness at the periphery and several irregular deeply stained bodies in the center. Twenty neuroglia cells in all are seen in this plate. Plate V. Fig. 14, is a photomicrograph of the same magnification,—1,400 diameters,—as the preceding and taken from a section of the same block. The section of the same form and size as that seen in Plate III. Fig. 7, is ten microns in thickness, and stained with methylene blue instead of methylene violet, but otherwise treated in exactly the same way. One noticeable point about this plate is the absence of neuroglia cells, in marked contrast to Plate IV. Fig. 12, indicating again that the neuroglia cells do not retain the methylene blue stain with the same intensity as when similarly treated with methylene violet. Here is seen a group of two Betz cells, the upper one, at this plane broadly pyramidal in shape, and the lower one presenting an irregular spindle shape. This latter, however, is only the base of the cell, the process above and to the left and that below and to the right being basal dendritic processes, and the apical dendritic process extending upward beyond the base of the upper cell in a different plane. There are two other Betz cells just above and slightly to the right of these cells in the section, not shown in the plate, thus making the group consist of four cells in all. Both of these cells are cut in the plane of the nucleus and large, rounded, deeply stained nucleolus. The nuclear body also contains some chromatic substance arranged in an indefinite network. At the base of the lower cell and extending into the large basal dendrite to the right as far as it is seen in this section is a distinct mass of yellowish pigment displacing the other cell contents. In the plate it is of a lighter color than the rest of the cell. Above this in the cell-body about the nucleus, and extending into the other basal dendrite, are numerous rounded and linear chromophilic bodies. The upper cell shows three dendritic processes going off from the base of the cell-body on the right side, and two from the left side. These processes and the large apical process contain mostly linear-shaped chromophilic bodies, arranged in distinct striæ, parallel to

the contour of the same. This arrangement also holds good at the periphery of the cell-body, the striæ there too being parallel to the contour of the same, but about the nucleus the chromophilic bodies are so closely packed that it is more difficult to make out this parallel arrangement. There is no pigmentary deposit in this cell. No neuroglia cells are to be seen in this plate, the methylene blue stain being used as stated above. Plate V. Fig. 15, from the same region and under the same magnification, 1,400 diameters, from a section ten microns in thickness and stained also with methylene blue, is an exceptionally fine photomicrograph of one of these

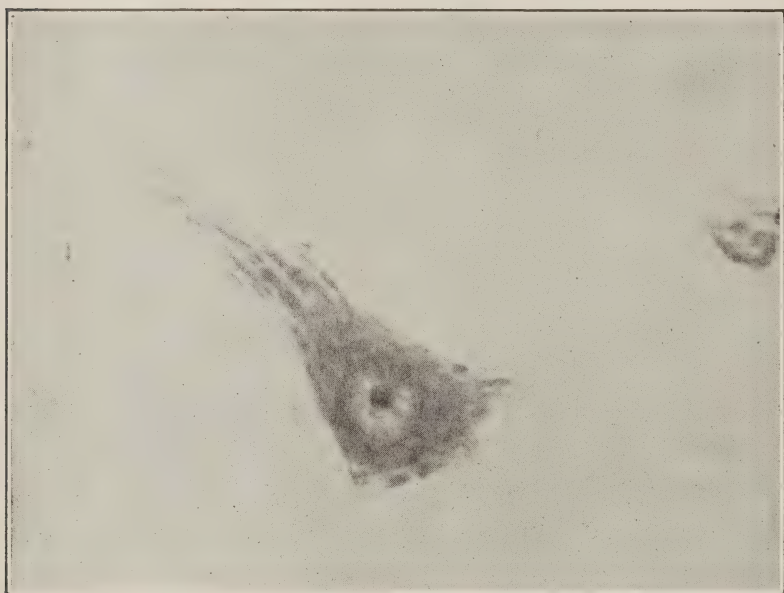


Plate V, Fig. 15

Betz cells, inasmuch as it shows the processes, especially the apical process, as far as the edge of the plate. It is usually difficult to obtain such an extent of surface of a cell in the same plane under such high magnification even when the microtome cuts in exactly the plane of the long axis of the cell. These dendritic processes are seen to be of no mean size, especially the apical process and the chromophilic granules extend far up into the same. Three processes are seen given off from the base, one at each angle, and the third descending from the center. The nucleus is distinct, centrally located, and contains a large nucleolus surrounded by an irregular chromatic network. The chromophilic granules are

larger and smaller rounded and irregularly spindle-shaped granules in the triangular-shaped body of the cell, approaching to a more linear form in the processes. This is a stichochrome nerve cell of the somatochrome group.

Turning again to Plate III. Fig. 8, the third layer will now be described. This is but .70 mm. in thickness as compared to 1.20 mm. for the same layer in Plate II. Fig. 3. The nerve cells in this layer are similar in form and structure to those found in the above mentioned plate. The neuroglia cells are much more numerous in all the layers than in Plate II. Fig. 3, due in large part, the writer believes, to the difference in the staining method. Capillary vessels, showing the walls made up of a single layer of nucleated flattened cells, are seen in several places, especially in the lower layer. Counts were made of the nerve and neuroglia cells in different regions in the manner previously described, using the same method and magnification. Counts of the nerve cells in eight different fields from various portions of the second and third layer were made, each field containing thirty-six squares, so that 288 squares were examined. There were found to be an average of .95 nerve cells to each square millimeter of the ocular net-micrometer, which multiplied by 100 gives 95 as the average number of nerve cells found to the square millimeter in the second and third layer of the cortex in this section. The first layer contained so few scattered nerve cells that no field was included for this portion of the cortex, as some fields here would contain no nerve cells whatsoever and would thus greatly reduce the general average where the Nissl stain is employed. On this account the nerve cell counts in this and all subsequent sections, as well as that of Plate II. Fig. 3, include only the second and third layers of the cortex. The nerve cells are thus fewer in number than in Plate II. Fig. 3, but they average larger in size, especially in the region of the large pyramidal and Betz cells; thus the frontal convolutions seem to contain more nerve cells than the central convolutions.

Neuroglia cell counts were made from three fields of thirty-six squares each or 108 squares in all from all three layers of the cortex. The average number of neuroglia cells was found to be 2.93 for each square millimeter of surface of the ocular net-micrometer, which multiplied by 100 gives 293 as the average number of neuroglia cells in each square millimeter of surface of the cortex from this section. The number in each square here varied from none in some squares to fourteen in one of the squares. The next highest number was eight, several contained seven, but most of them contained from one to four. Counts were also made from a section of the same thickness, ten microns, and from the same block as the preceding, but stained with methylene blue instead of methylene violet. With this exception the technique was ex-

actly the same in the two preparations. Counts were made from six different fields of thirty-six squares each, and the average number of nerve cells to each square mm. of the ocular net-micrometer from various parts of the second and third layers was .94. This result multiplied by 100 gives 94 as the average number of nerve cells to each sq. mm. of surface of the section as compared to 95 in Plate III. Fig. 8, a remarkably small difference of only one cell to each square millimeter, both counts being made, the one entirely independent of the other. Counts were made

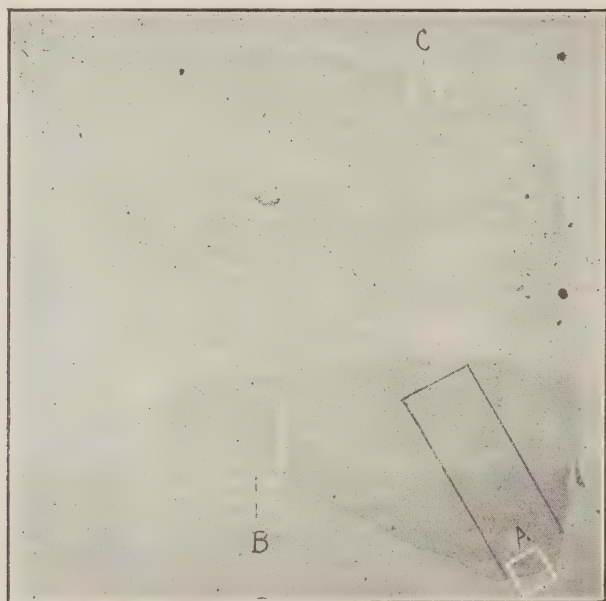


Plate VI, Fig. 16

from seven different fields of 36 sq. mm. each from various parts of all three layers of the cortex to determine the average number of neuroglia cells. The average number to each square millimeter of the ocular net-micrometer was found to be .84, which multiplied by 100 gives 84 as the average number of neuroglia cells to each square millimeter of the section as compared to 293 in Plate III. Fig. 8, where the methylene violet stain was the stain used. This further confirms the statement previously made that methylene violet has a greater affinity for these neuroglia cells and the latter retain this dye much more tenaciously than they do the methylene blue in the subsequent differentiation, whereas the nerve cells retain either dye about the same. The same fact is well

brought out in Plate IV. Fig. 12, and Plate V. Fig. 14, previously described. In the latter two, Betz cells are seen under a magnification of 1,400 diameters. The stain used here is methylene blue, and no neuroglia cells are to be seen. In Plate IV. Fig. 12, of the same magnification, but stained with methylene violet, twelve of these neuroglia cells are seen in the immediate vicinity of this large Betz cell. Sections were also prepared from blocks taken from the middle and lower thirds of this same convolution,—the anterior central,—and it was found that the general type of cortical lamination and histological structure remained the same. Plate VI. Fig. 16, is a photomicrograph of a section taken from a block including the entire anterior central convolution for a distance of .4 mm. in its lower third and is representative of the entire lower portion of this convolution. The shape of the convolution is seen to have undergone considerable modification as compared to the upper third, seen in Plate III. Fig. 7. This section is ten microns in thickness, fixed in alcohol 95 per cent., stained with methylene violet, the other technique being similar to previously described sections. The segment of cortex shown in Plate VI. Fig. 17, under a magnification of 100 diameters, was taken from the point indicated by the ink lines here and opposite *a*. It is seen from the mechanical arrangement of the cortex to be at the most acute angle and where the same is thickest. At the opposite less acute angle the cortex is not quite so thick, and intermediate between these angles, and also on the anterior and posterior surfaces the cortex is still thinner. It will be noted that the depth of the sulcus anteriorly at *c* where it passes over into the third frontal convolution and is thus adjacent to the motor speech area, is more shallow than the posterior sulcus or fissure of Rolando. The first layer is irregular in depth, somewhat thicker at the angle opposite *a*, and still thicker at *b* and *c*, whereas at the vertex it is thinner. The general radial direction of the cells from the medullary central white substance to the surface is fairly well seen here. In the second layer the most noticeable feature is the entire absence of the Betz cells which are so marked in the upper portion of the convolution. Some very large pyramidal cells, intermediate in size to the Betz cells and the ordinary larger pyramidal cells, are to be seen in this layer however, as will be noted in the following (Plate VI. Fig. 17): This layer merges into the third or irregular cell layer, which latter is gradually lost in the white central substance of the gyrus. At the vertex in the cortical layer the section is cracked; this being an artefact resulting from manipulation. The outer layer is seen to be also torn in places from the same cause. Plate VI. Fig. 17, is taken from the strip indicated in ink in Plate VI. Fig. 16, and is magnified 100 diameters. The first layer is .25 microns in thickness and similar in structure to the same layer in the

upper portion of the convolution already described. In the second layer, however, the greatest difference is found. The large Betz cells practically disappear in the lower third of this convolution, although large pyramidal cells simulating them in general structure, but lacking the size, are found in the middle portion of this layer. In this lower third of the convolution the second layer as seen in this section is 1.50 mm. in thickness, and the large pyramidal cells are seen arranged in irregular groups in its middle portion. Below them is seen a region *a* averaging .25 mm. in thickness in which small pyramidal cells are almost exclusively found. Below this larger pyramidal cells are seen, but not in great numbers. The division between this and the lower or spindle cell layer is not well marked, the latter measuring .90 mm. in thickness. The cells here are irregularly polygonal or spindle shaped and do not differ from those in the same region in the upper part of this convolution. The total thickness of the cortex at this point is 2.65 mm., exactly the same as of the section shown in Plate III. Fig. 8. Counts of the nerve and neuroglia cells were made in this region in exactly the same manner as in the previously described regions. This section, as above stated, is ten microns in thickness and stained with methylene violet and in seven fields of 36 sq. mm. each from various portions of the second and third layers, the average number of nerve cells to each square millimeter of surface of the ocular net-micrometer was found to be 1.08, multiplied by 100 gives 108 as the average number of nerve cells to each square millimeter of surface of the section in the second and third layer. The nerve cells in the first layer were so few and scattered that no counts of the same were made. In eight fields of 36 sq. mm. each from various portions of the three layers of the cortex in this section the average number of neuroglia cells to the square millimeter of surface of the ocular net-micrometer was 2.48, multiplied by 100 gives 248, as the average number of neuroglia cells to each square millimeter of surface of the cortex of this section. Here again it will be seen that the methylene violet stain shows a larger number of neuroglia cells—248, while the nerve cells—108, are but slightly in excess of the average number in the two previously described sections of the upper portion of the anterior central convolution, and slightly less in number than in the sections described from the frontal convolution (Table II.).

Passing now to the region posterior to this, we come to the posterior central convolution of this central or motor region. This posterior central convolution is regarded as a transition region between that anterior to the fissure of Rolando, or motor region, and that posterior to the same region, or sensory region of those authors who divide the cortex into a sensory and motor type of cortical lamination. The first and third layers here are prac-

tically the same as in the region anterior to the fissure of Rolando. The second layer, however, lacks the regularity of the corresponding layer in the above mentioned region, but as will be seen in the plate (Plate VII. Fig. 19), there is nothing new or different in this layer from the corresponding layer in the plates already described. The large Betz cells seen only in limited portions of the region just anterior to this and contained in but a very small portion of this so-called motor region are replaced here by some smaller sized, irregularly situated pyramidal cells, singly or in groups, and of similar internal structure, and mostly to be found

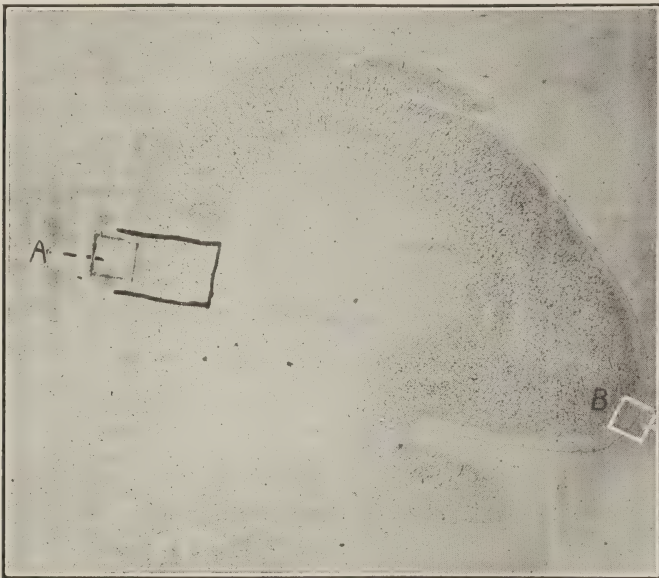


Plate VI, Fig. 18

in the middle and lower portion of the layer. As typical of this posterior central convolution, a section was taken from a block including the middle portion of this convolution in its entirety for a distance of 4 cm. (5, Plate I. Fig. 1). The section is 6 2-3 microns in thickness, fixed in 95 per cent. alcohol, and stained with methylene blue, technique otherwise similar to all other sections. Plate VI. Fig. 18, is a photomicrograph of this section, magnified 14 diameters. The convolution here presents a peculiar mechanical formation, causing a great variation in thickness of the cortex in different portions, being over twice as thick at *b* as at the point *a*, from which latter point Plate VII. Fig. 19 was taken. This condi-

tion is not an artefact, as under high magnification no indication of disturbance of the relations of the cells is to be observed. At the vertex, however, there is an artificial break in the first and upper part of the second layer at two points. The first layer is seen to vary in thickness, being thicker at the acute angle below *b*, and gradually becoming thinner as it approaches the vertex, to become again thicker below *a*. The general radial direction of the cells is well seen, especially at the vertex and anterior aspect, as at *a*. At *b* it is not quite so well made out, but exists to a fairly well marked degree when examined under a higher power. Pyramidal cells of considerable size are seen in some parts of this layer, more especially in the middle portion, but in no case do they approach in size to the Betz cells, nor are they for the most part situated in the same portion of the cortex. The third layer is thin and soon lost to view in the medullary white substance. Plate VII. Fig. 19, is a photomicrograph of the strip *a* enclosed in ink lines in Plate VI. Fig. 18, and magnified 100 diameters. The first or upper layer at this point is somewhat irregular in contour and averages .20 mm. in thickness. It contains neuroglia cells and some scattered nerve cells similar in appearance and structure to those described in Plate II. Fi. 3, for the same region. The second or pyramidal cell layer is especially interesting, owing to the irregularity of the distribution of its cells. At the upper portion the small pyramidal cells are seen closely aggregated together. Below this these cells become more scattered, and irregularly interspersed among them are medium sized and larger pyramidal cells. Some of the larger of these have a tendency to be arranged singly or in groups and have an internal structure similar to the giant pyramidal or Betz cells found in parts of the anterior central convolution and in the posterior part of the first frontal convolution. The chromophilic granules are, however, of smaller size though similarly arranged in some of the cells. About the middle of this layer, opposite *a*, is seen another region in which the small pyramidal cells predominate, and in which there are scarcely any larger pyramidal cells. Just below this again are seen large pyramidal cells arranged singly and in groups, forming the lower portion of this second layer which measures about 1.45 mm. in thickness and below gradually merging into the third or spindle cell layer. In addition to the spindle cells there are many irregular polygonal cells founds in this layer which at this point is about .60 mm. in thickness and only partially shown in the plate; the upper two-thirds being seen here. The lower third, not shown, is similar and gradually merges into the white medullary substance. The entire cortex is thus 2.25 mm. in thickness at this point, whereas, as above mentioned at point *b* in Plate VI. Fig. 18, it is over 5 mm. in thickness. Taking into consideration the variations found in different parts of the same area, often quite near

together, and considering the general type of arrangements, it does not seem to the writer that this area varies to a very marked degree from the general type, or at least to such an extent as to be especially marked off from the other regions of the cortex. There is certainly a greater variation between different regions of the anterior central convolution as in the upper and lower portions, where some parts have no Betz cells and others have them variously distributed, as well as other differences, than between this region and the frontal region as a whole. Those who separate the cortex into two types,—an anterior or motor type, and a posterior or sensory type,—regard this convolution as the transition region between the two. An examination of sections, both anterior and posterior to this convolution, shows that the same general plan of arrangement is carried out in all parts of the external surface of the cortex with only minor differences which do not seem sufficient to make such a marked distinction applicable anatomically, and certainly not histologically. The size and form of the cells and their general arrangement differs but slightly from that of the region anterior and posterior, with the exception of the Betz cell groupings in localized portions of the former.

Nerve cell and neuroglia cell counts were made with the ocular net-micrometer here in this region in the same manner as in the other previously described sections. Eight fields of thirty-six square millimeters each, from various portions of the second and third layers of the cortex, were examined and 185.5 nerve cells were found to be the average number for each square millimeter of surface of the section. In the same manner the neuroglia cells were counted in eight fields from all three layers of the cortex and 109.2 neuroglia cells were found to be the average number for each square millimeter of surface of the section in the cortex. Here methylene blue was the stain employed with the resulting low average number of neuroglia cells, whereas the average number of nerve cells is found to be higher than in any of the previously described sections anterior to this region, and it will be seen subsequently that it is also higher than that found for the region posterior as well although the section is 6 2-3 microns in thickness, and some already described are 10 microns in thickness (Table II.). This, however, bears out the statement previously made that sections between six and ten microns in thickness contain practically all nerve cells that can be seen in one plane.

Parietal Region.—Posterior to the region just described we come to the parietal region, made up of the superior parietal, supra-marginal, and supra-angular convolutions. The general arrangement of the layering of the cortex is fairly uniform throughout this region and the section represented in Plate VII, Fig. 20, from a block taken from the supra-marginal convolution is typical of this region (7, Plate I, Fig. 1). The block was

fixed in Van Gehuchten's fluid, stained with methylene violet, and other technique as for previous regions. The actual size of this section is 7 mm. in its greatest width, and 8 mm. in length, and it is 6-3 microns in thickness. As seen in the photomicrograph, magnified 14 diameters, the convolution is wider above at the surface than deeper down in the sulcus. At the point *a* is seen the position of the strip from which the photomicrograph represented in Plate VII, Fig. 21 was taken. At *b* and *c*, owing to the

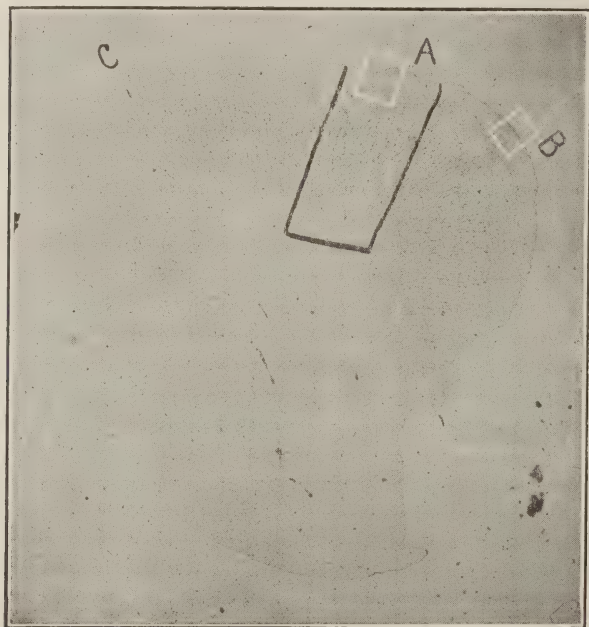


Plate VII, Fig. 20

greater angle, the cortex is considerably thicker than at *a*. The shape of the convolution here is seen to be different again from any of the preceding, thus giving a somewhat different mechanical arrangement, with a broad flattened vertex and shorter anterior and posterior aspects. At the lower portion, on the right, a part of the cortex was cut away in removing the block. In the vicinity of *a* there are some minute artefacts, causing an irregularity of the surface here, and small fragments are partially torn away at two points. The first, or superficial layer, is fairly uniform in thickness, excepting opposite *a*, which will be discussed more fully in describing the next plate (Plate VII, Fig. 21), and upon



Plate IX, Fig. 25.

late XV, Fig. 45.

Plate XV, Fig. 47.



Plate IX, Fig. 25.

Plate XI, Fig. 31.

Plate XI, Fig. 33.

Plate XIV, Fig. 43.

Plate XV, Fig. 45.

Plate XV, Fig. 47.

the posterior and anterior aspects, where this layer is slightly thicker. The radial arrangement of the cells of the second layer is well shown, and the cortex is seen to be thicker at the angles *b* and *c*, as before mentioned. Throughout the middle and lower part of this layer well developed pyramidal cells are seen, some almost approaching in type small Betz cells. The third layer presents nothing unusual, and gradually passes over into the white medullary substance. Plate VII, Fig. 21, shows the strip indicated in ink opposite *a* in Plate VII, Fig. 20, under a magnification of 100 diameters. The first or superficial layer is seen to be irregular at this point, about 20 mm. in thickness at the right of the plate, then increasing rather suddenly to .25 mm. for a short distance, when the outline suddenly descends, decreasing the layer at this point to .15 mm. It then gradually increases until, at the left of the plate, it is .22 mm. in thickness. The extreme thickness near the center, although the surface is only slightly torn at several points and scarcely noticeable, yet has the appearance of an artefact, due no doubt to the unequal pressure in the manipulation of removal, when the brain was in its natural soft condition, prior to any post-mortem changes. A short distance below the surface at this point the cortical substance itself is seen slightly broken, thus confirming the artificial nature of the irregularity and increase in depth. The nerve and neuroglia cells are similar to those found in this layer in the regions anterior to this. The second or pyramidal cell layer is 1.60 mm. in thickness, and contains small pyramidal cells exclusively in the upper portion. Very soon, however, larger pyramidal cells appear, and at about the middle of the layer are the greatest in size and most numerous of the layer. Some of these large pyramidal cells in point of size and structure appear intermediate between the large and giant pyramidal or Betz cells. They also tend to be arranged singly or in groups, with smaller cells about them. Just below the middle of this layer, at *a*, is found a region .20 mm. in thickness, conspicuous by the almost entire absence of large pyramidal cells, and made up for the most part of small pyramidal cells. Below this large pyramidal cells again appear, intermingled with the small pyramidal cells, and the layer gradually passes over into the lower or spindle-cell layer. The pyramidal cells have the same internal structure as those in the region anterior to this, in the largest ones the chromophilic granules simply being larger and more conspicuous; thus these cells come under the same classification, according to Nissl, as those in the frontal and central convolutions. The third or spindle-cell layer is .70 mm. in thickness, and not distinctly separated from the layer above. The arrangement of the cells and their form and size, as well as the internal structure, is in no way different from the corresponding layer in the cortex anterior to this. The total thickness of the cortex here

is then 2.50 mm., being .25 of a mm. thicker than in the section of the posterior central convolution previously described, .15 mm. less in thickness than the section shown in Plate III, Fig. 8, from the anterior central convolution, and .35 mm. less than in the section of the first frontal convolution shown in Plate II, Fig. 3. The variation in thickness is thus seen to be slight and no greater, and even not so great as can be found in contiguous portions of any one convolution. In other parts of the parietal region the arrangement of the cortical lamination is similar to that described above with minor modifications, due to differences in shape, etc., of the convolutions. Here nerve cell and neuroglia cell counts were made in the same manner as in previous regions, and the average number of nerve cells to the square millimeter of surface of the section was found to be 104, whereas the average number of neuroglia cells for the same area of surface of the section was 180.90. This is the lowest average of nerve cells in any region of this brain, with the exception of the upper portion of the anterior central convolution. The methylene violet stain makes the average number of neuroglia cells quite high, however, though considerably less than in the upper portion of the anterior central convolution.

Temporal Region.—Below the parietal region just discussed we come to the three temporal convolutions—the first, second and third. Here the general arrangement of the cortical laminations and the cell structure is practically the same in the three convolutions, and bears a striking resemblance to that found in the frontal region. A block was taken from the anterior portion of the first temporal convolution near the apex (6, Plate I, Fig. 1), the entire width of the convolution and for a distance of .4 cm. The actual size of the section from this block represented in the photomicrograph (Plate VII, Fig. 22) is 8.5 mm. at the widest point, and 8.5 mm. in length on the right, and 6 mm. in length on the left, and it is 6-2-3 microns in thickness. The block was fixed in Van Gehuchten's fluid, stained with methylene blue, all the other details of technique being similar to that employed for previously described sections. This photomicrograph is magnified 14 diameters, and here the shape of the gyrus is seen to be somewhat similar to that in Plate VII, Fig 20, but broader above, and also with a broader base. The vertex is flattened similarly, resulting in the more acute angle at *b* and the slightly obtuse angle at *c*, at which points the cortex is thicker than at the vertex or the anterior or posterior aspects. The cortex is seen to be uniformly thicker along the surface, opposite *d*, than upon the opposite aspect at and above and below *a*. The first or superficial layer is thicker at *c* than in other portions, and is somewhat broken in places along the vertex. The second layer varying in thickness, being thickest at the points mentioned above, where the entire

cortex is thickest, shows the radial arrangement of the cells very well, especially at the vertex and opposite *a* and *b*. The larger nerve cells appear quite conspicuous in the middle and lower portion of this layer in places. The third layer is quite uniform, disappearing in the white medullary central substance. Plate VIII, Fig. 23, shows the strip *a* of Plate VIII, Fig. 22 at a magnification of 100 diameters. Here the differentiation has been carried to about the same stage as in Plate II, Fig. 3, and the paucity of neuroglia cells is equally as apparent. Again the general arrange-

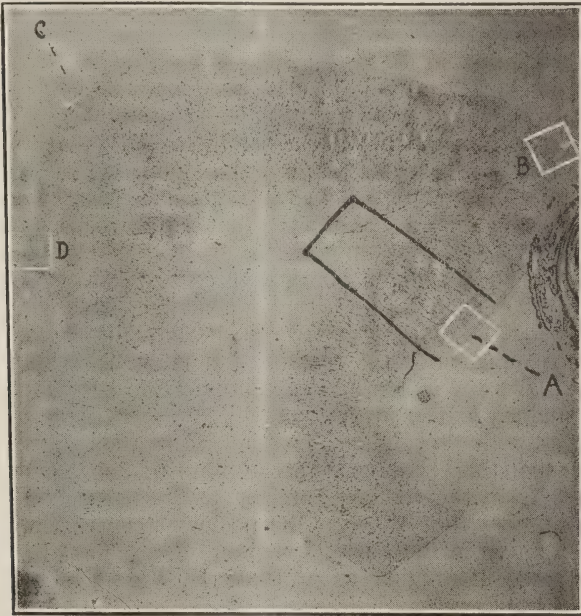


Plate VII, Fig. 22

ment of the cells in the two plates is remarkably similar. The first or superficial layer is here .25 mm. in thickness, and contains, for the most part, neuroglia cells and a few scattered nerve cells. The second or pyramidal cell layer measures 1.65 mm. in thickness, as compared to 1.40 mm. in Plate II, Fig 3, being thus slightly thicker than in the latter. The cells are arranged practically the same, however, the small pyramidal cells above intermingled with larger pyramidal cells in increasing numbers deeper down in the layer. Here again, at a point somewhat deeper than in Plate II, Fig. 3, is to be found a region at *a*, of the same thickness—.30 mm., where small pyramidal cells are found almost ex-

clusively, followed again by a region containing large pyramidal cells in considerable numbers in addition to small pyramidal cells. This layer finally passes over into a narrow (.40 mm. thick) third or spindle-cell layer. This layer is also similar to that of Plate II, Fig. 3, with the exception of its depth, the latter having a spindle or irregular cell layer, measuring 1.20 mm. in thickness. As will be seen by referring to Plate I, Fig. 2, the strip *a* represented in Plate II, Fig. 3, was taken from the point of greatest curvature, where the cortex is usually the thickest, whereas Plate VIII, Fig. 23, was taken from the point *a* shown in Plate VIII, Fig. 22, from the side of the section where the cortex is thinner. The entire depth of the cortex is found to be 2.30 mm., as compared to 2.85 mm. in Plate II, Fig. 3, a difference of .55 mm. There are no larger pyramidal cells here intermediate in size between the Betz cells and the ordinary large pyramidal cells, as were noted singly or in small groups scattered in the central and parietal convolutions already described, and, as we shall see, also occur in the occipital convolution. The internal structure of these cells and their classification are similar to that for the cells described in Plate II, Fig. 3. Nerve and neuroglia cell counts were made from this section, which, as above mentioned, is 6 2-3 microns in thickness and stained with methylene blue. Seven fields of 36 sq. mm. each gave an average of 146 nerve cells to each square millimeter of surface of the section, and eight fields of the same size gave an average of 131.90 neuroglia cells to each square millimeter of surface of the section. It will be seen upon comparing these figures with those from sections of regions more anterior and above, that they show a larger number of nerve cells than in the sections from the regions anterior to the fissure of Rolando, but a less number than that for the posterior central convolution, and more than from the parietal region. The neuroglia cells are about the same in number as in Plate II, Fig. 3 of the frontal region.

(*To be continued.*)

Periscope.

ARCHIV FÜR KRIMINAL ANTHROPOLOGIE

(Vol. 12, 1903, Nos. 2, 3.)

1. Daktyloscopie. C. WINDT.
2. Rendering Latent Impressions Visible. F. PAUL.
3. Impressions of Elmira. WINTRY.
4. Medical Experts in Psychiatry. J. BERZE.
5. Infanticide by Cold Bath. R. MOTHES.
6. Alcoholism and Testimony. R. POLLITZ.
7. Signs of Death by Hanging. STRASSMANN.
8. Falsification of Seals. W. SCHÜTZE.
9. Preliminary Cross-Examination. H. GROSS.
10. Anatomical Seat of Criminal Tendencies. P. NÄCKE.
11. Legal Views of Crime. R. MOTHES.
12. Reform Movement in Criminal Procedure. G. LELEWER.
13. Legal Science in Greenland. D. B. OEFELE.
14. Reading of Thoughts. H. SCHNEICKERT.
15. Superstition: Fortune-Telling and Quackery. W. SCHÜTZE.
 1. *Daktyloscopie.* C. Windst points out the great value of the papillary lines at the finger tips for identifying criminals. The pattern remains the same throughout life, and even if a portion of the skin is torn off accidentally, the new-formed skin will not alter the design. Even when bodies have been lying in water for weeks, they can be readily identified by impressions from the fingers. All patterns may be divided into four classes, which have received the following names: Bow (arcus), loop (lasso), whirl and combined pattern. A very extensive system of cataloguing the impressions from all ten fingers by means of arbitrary letters and fractions is described in detail. Compared with anthropometry, the method seems to be equally reliable and much simpler; it can be applied just as easily to children, females and cadavers. Occasionally the criminal himself helps in his identification by leaving behind an impression of his fingers on glass, etc. By suitable methods these impressions can be rendered more visible and permanent.
 2. *How to Render Visible Latent Impressions.* From F. Paul's article it appears that many chemicals can be used to render visible and permanent accidental impressions of fingers on glass, paper, etc. The simplest method is to expose the object to the fumes of iodine. Silver-nitrate in 8 per cent solution also gives good results if the paper is then exposed to sunlight and treated with hyposulphite of soda. Other available substances are ink, many powders in moderate subdivision (calomel), permanganate of potash. With glass, the fumes of hydrofluoric acid may also be employed.
 3. *The Reformatory in Elmira.*—Wintry has visited Elmira and speaks very favorably of the management of the institution and the régime among the inmates. He believes that from 75 to 80 per cent of these are completely cured.
 4. *Difference of Opinion Among Experts in Psychiatry.*—J. Berze states that it is oftentimes impossible to estimate correctly the mental capacity of a patient. This refers especially to those cases which are not really mentally diseased but merely exhibit a deficient mental development. The individual equation plays a prominent part here, since we possess no normal

standard by which to gauge the degree of intelligence. Even in acquired conditions there may be a psychical weakness rather than definite and characteristic symptoms, so that the difference from the normal is quantitative rather than qualitative. Almost insurmountable difficulties may be encountered in distinguishing between temporary and permanent aberrations in neurasthenia, hysteria and epilepsy. Last, but not least, the expert will often be in a quandary to express an opinion in psychopathic deficiency, that intermediary condition which has underlying it neither a normal nor an abnormal mind. There is no proper place for these patients as yet; they belong neither in an insane asylum nor in prison, yet the expert is often forced to choose between the two. It is difficult to find a remedy for the difference of opinion so frequently encountered; even if separate institutions existed for these patients there would still be occasion for dispute, but the consequences would not be so serious.

5. *Attempt at Infanticide by Means of a Cold Bath.*—R. Mothes briefly records the case of a woman who had purposely left her infant in a cold bath. The court acquitted the woman of attempted murder, but recommended her punishment for the affliction of bodily injury which might have resulted fatally.

6. *Testifying Upon Alcoholic Disturbances Before Court.*—Pollitz relates how difficult it may sometimes be to properly class alcoholic mental disturbances with reference to the ultimate psychical derangement which accompanies them. There is a tendency on the part of the laity to look upon alcoholism more as a moral defect than a disease, and, in consequence, the judge and jury are frequently inclined to condemn too harshly. The matter is frequently complicated by the fact that it is difficult to make the proper diagnosis, especially if the expert sees the patient after weeks or months of enforced temperance. Alcoholic psychosis may be divided into four classes, viz.: Delirium tremens, acute hallucinatory insanity, the acute pathological intoxications, and, lastly, chronic delirium. Of these, delirium tremens forms the prototype; it is that peculiar condition where the afflicted is fully aware of his own condition, but mistakes completely his surroundings. Prominent symptoms are optical delusions, marked suggestibility and motor unrest and a good recollection of the details of previous attacks. In the second type the delusions are more markedly of an acoustic character, the patients are better oriented, and the entire course is more chronic. Pathological intoxications are accompanied by a clouded consciousness. The recollection of the details is almost entirely lost, and sleep always follows. Illustrative cases of these three conditions are given.

7. *Signs of Death by Hanging.*—Strassmann had occasion to do an autopsy on an old man who first attempted to hang himself and then fired a bullet into his brain. As in a previous case recorded, tears in the mucous membrane at the laryngeal orifice were found.

8. *Methods Employed in the Falsification of Seals.*—A detailed article by W. Schütze, stating how signatures, seals, etc., are commonly altered by criminals.

9. *Value of Preliminary Cross-Examination.*—This article of H. Gross is directed against the suggestion to do away with the customary preliminary cross-examination. When properly conducted by an experienced judge it is the most valuable of the three commonly employed in Germany.

10. *The Anatomical Seat of Criminal Tendencies.*—P. Näcke does not believe with Lombroso that criminals and epileptics possess a prominent median occipital groove secondary to hypertrophy of the worm of the cerebellum. Prominent anatomists have shown that this condition is found almost as often in the brains of normal individuals (in 2 to 3 per cent), and hence cannot be looked upon as an atavistic change. Despite the many writings of Lombroso, the real anatomical seat of criminal tendency is as unknown to us as it ever was.

11. *Incorrect Legal Views as a Cause of Crime.*—R. Mothes mentions several peculiar notions on legal points prevalent especially among the lower classes. They often constitute reminiscences of former laws, and not infrequently lead to or justify crime.

12. *The Reform Movement in the Trial of Criminals.*—G. Lelewer discusses the contemplated reform in criminal practice, and advises against too great leniency.

13. *Legal Science in Greenland.*—The inhabitants of Greenland still possess very primitive ideas concerning law. According to D. F. B. Oefele, murder is very rare since the national character is peaceable and abhors dispute. A common practice, however, is to hasten the death of the old and dying. Married life is rather loose, and it is considered more of an advantage than a disgrace to possess children born out of wedlock. Disputes are usually settled by means of the so-called "drum dance," where the one who makes most noise is generally the victor.

14. *Reading of Thoughts.*—H. Schneickert has been present at several seances and has found that the thoughts to be transmitted are always communicated to the medium by a carefully studied and often complex system of signals. These are passed at moments when the attention of the public is diverted. Criminals frequently have a similar mute language, and partners should always be kept at a distance from each other, and all motions of their hands, face, etc., should be carefully watched.

15. *Superstition, Fortune-Telling and Quackery.*—The superstition of the lower classes has frequently ruined reputations and destroyed family ties. In the case cited by W. Schütze a perfectly innocent man was accused by almost all his acquaintances of arson because a "wise man" had thrown suspicion upon him. It is often very difficult to punish these quacks. In this case it took one and a half years to gather sufficient evidence.

O. HENSEL (New York).

ARCHIVES DE NEUROLOGIE

(Vol. 15, 1903, No. 89, May.)

1. A Study of Mania. SOUKHANOFF—GANNOUCHKINE.
2. New Observations on the Neurosis of Anguish. P. HARTENBERG.
3. Physiopsychology among Religious Orders. BINET—SANGLÉ.
4. Critical Review of Stigmata of Degeneration.

1. *A Study of Mania.*—Drs. Soukhanoff and Gannouchkine of University of Moscow founded their study upon material accumulated in the psychiatric clinic of Moscow. The authors have been able to classify the "observations" of all patients, both interne and externe during fifteen years from 1887 to 1902. The total number of patients were 4,434, of whom 2,840 were men and 1,594 were women. Among these, they recognize mania in but 40 cases, of whom 16 were men and 24 women. In the category of mania, the authors exclude from consideration all cases where, before or after the onset of mania, a depressive state was noted; these they classify as circular psychosis. All cases are also excluded where mania appears as a complication of general paralysis, a senile dementia, etc. "The above data indicate very clearly that mania is a very rare psychic malady."

Women are prone to suffer from mania more often than men, and if the number of male and female aliénéés were equal, the ratio of men to women affected with mania would be as 1 to 2.68. Melancholia, particularly in women, appears as one of the most frequent of mental maladies; occurs almost seven times as often as mania. These views, they say, are those of other authors. Figures show that mania manifests itself most often between the ages of 16 and 25 years. The age of earliest access, 13 years; latest at 66 years.

2. *New Observations upon Neuroses of Anguish.*—Hartenberg, after

Freund of Vienna, and upon his own studies, classifies the subject: (1) General irritability, including often auditive hyperesthesia; (2) A state of habitual anxious expectation of some evil which threatens; (3) Crises of acute agony, beating of the heart, etc.; (4) Equivalents of crises, represented by nocturnal fright, with distressing dreams, etc., variations of general nutrition, with periodic fattening or emaciation; (5) Phobias and obsessions.

The first observation cited is that of a married woman, Madame L., twenty-six years of age, mother of a child of four and one-half years. She was in great mental distress, the origin of which was as follows: While absent from her home, "a friend of the house, made courtship to her and engaged her in a flirtation sufficiently advanced. There was great reciprocal attraction; the situation became perilous for the honor of the young woman, when suddenly the gallant friend, seized with remorse, put an end to their relations. They almost ceased from seeing each other." This, instead of calming her for the future, induced bitter remorse. She was in continued inquietude. A thousand questions arise in her mind. "Does she still love her husband? Her child? She is a great criminal. She ought to be punished. What misfortunes will happen to her, how escape them?" She weeps, is in despair, and calls for death to deliver her from her martyrdom. Somatic troubles accompany the mental state, etc. The author institutes a treatment tonic and reconstituent. While she improves in bodily health, sleeps, etc., the obsession, fear of punishment with scruples, reproaches, ideas of suicide, continue, and after six months the mental troubles have subsided but not disappeared. The neurasthenia is rapidly cured; the neurosis of agony is not amended till much later.

The author next propounds, with qualified approval, an hypothesis of Freund that neuroses of anxiety are caused by insufficiency of satisfaction of sexual want. He reports in his own practice the case of a widow, who afterward remarried, which gave some evidence of Freund's opinion. He also reports in much detail a case of the neurosis of anxiety produced by the emotions of automobilism. In 1899, M. F. commences automobiling, at first with a tricycle, then with a voiturette, and lastly with a carriage of six horse power. In May, 1899, he journeys from Nice to Macon, with four halts. The first three days pass without incident in spite of the great fatigue produced by the close attention required in guiding the carriage. The fourth day, in getting out at Neufchateau for luncheon, he finds himself greatly indisposed. He experiences severe pain in the epigastric region, like that in extreme emotion. His teeth chatter, he shivers, has violent beating of heart, and is assailed with fear of sudden death. In October following, after three days' journey in the automobile, he dines with good appetite and sleeps at Avignon. Towards eleven o'clock he is awakened with an atrocious terror. It seems to him that it is the end, that his heart will break; that he will instantly die—all this subsides in half an hour. A medical consultation reveals no heart lesion, and attributes the symptoms to a gastric state. M. F. has given up the automobile. *A propos* of this: on page 480 of this number, there is a report of a meeting of the Society of Hypnology and Psychology, at which there were remarks upon the psychology of speed.

HACHET-SOUPLET. Whoever is in a situation to increase at will his speed is entirely carried away by the intense impressions which he experiences; he loses self-mastery; he is intoxicated; there are born in him feelings which he has not in a normal state; pride, combativeness, anger, hatred, spitefulness, violence. These feelings vary in form and degree, according as it concerns the driver of an omnibus, the coachman on his cab, the horseback rider, the automobiliste.

BERILLON. There is a great analogy between the euphoria of speed and that of morphine. Those who allow themselves to be carried away with

speed-making, for its own sake, without any end of utility, are most often degenerates, deprived of power of doing things in moderation, have lost empire over themselves and contend in a fury against the slightest obstacles.

PAUL MAGNIN. When we come to the question of speed, of tobacco or of alcohol, we have an affair of individuals, who in use, are unable to keep from abuse; are in effect deprived of all power of moderation, to the point of losing the instinct of self-preservation. One physician thought that speeding a horse involved a difference, as it was control of an intelligent being, to which replies.

LUX. In Algeria I have been a witness of Arab fantasias. They are demi-fools, intoxicated by the dust, transported by a savage fury; have no longer any sense of moderation. Their euphoria resembles that of automobilists, and the horses even are likewise seized with it.

The author states, in summing up, that we may consider the clinical syndrome of the neurosis of agony as real and generally accepted at this day. But it remains not less true that there are some cases, where the affection as described exists in a pure state, without any participation of neurasthenia or of melancholia, and which spontaneously or under the effect of care is cured entirely, without ever turning into asthenic neurosis or into psychosis of anxiety.

3. *Physio-psychology Among Religious Orders: Nuns of Port Royal.*—The author continues and concludes his studies of the seventeenth century monks and nuns of Port Royal, France. After giving minute biographies of several, he sums up by saying that he finds a neuropathic and religious heredity in one of the nuns and, in two, a religious heredity. Of three others, one was suffering from dysentery and suppurating hepatitis, and obtained relief in her troubles by auto-suggestion; the second was in general bad health; at twenty-seven had vomitings; at thirty-two, spitting of blood; the third also was in weak health, and died after five days of "oppression in the breech," with continuous fever. Of the four, of which we know the age at death, the first died at fifty-one, the second at nearly seventy-four, the third at sixty-eight, and the fourth at eighty; the average of the four was sixty-eight.

As to character, one passed as intelligent, another said she was "poor in spirit" (Query: Does the author know of the beatitudes?), two presented a predisposition to sadness, one to weakness and fear; all were of hypersuggestibility; three had a certain suggestive power.

4. *Stigmata of Degeneration.*—This number contains a review of certain articles by René and Henri Larger on the "Obstetric Stigmata of Degeneracy." Besides physical and mental stigmata there are also obstetric stigmata of degeneracy as seen in anomalies of gestation, such as sterility, twin-bearing, miscarriage, ectopic pregnancy, all the anomalies of the placenta, of the membranes, of the cord, all abnormal presentations, etc. These views are contested by Poiak and the accoucheurs: First observation—First husband normal; presentations normal in "OIGA"; second husband degenerate, presentation of the face. Second observation—First paramour normal, two successive presentations normal in "OIGA"; second paramour degenerate, presentation by the breech; the second paramour is a married man who has, simultaneously by his wife, a child born also by the breech. Third observation—Husband degenerate, three pregnancies with, each time, presentation abnormal with puerperal convulsions. The wife takes a paramour perfectly normal; three new accouchments, all normal in "OIGA," and without puerperal eclampsia.

RICHARDS, Amityville.

MONATSSCHRIFT FÜR PSYCHIATRIE UND NEUROLOGIE

(Vol. 13, 1903, No. 5, May.)

1. The Aphasic Symptom-Complex. E. STORCH.
2. Contribution to the Knowledge of Transcortical Aphasia. M. BERG.
3. Contribution to the Further Knowledge of the Richness of the Cerebral Cortex of Human Beings in Myelinated Nerve Fibers. E. L. F. S. BRÜCKNER.

4. The Pathology of Paralysis of the Peroneus. S. DAUS.

1. *Aphasia*.—Storch commences his article with a careful analysis of psychical perception. This is of psychological rather than of medical interest. He mentions, however, as one of the most important facts, that attention to any part of the body diminishes the way of irritability of the motor cortical cells that cover that part. Speech differs from the visual perceptions inasmuch that it is wholly dependent upon hearing. He analyzes the acoustic perceptions. He draws a sharp distinction between musical perceptions and phonetic perceptions, the two being entirely independent. (The paper is still unfinished.)

2. *Transcortical Aphasia*.—The essential symptoms of transcortical aphasia are paraphasia, paraphagia, and echolalia. The variation in the anatomical changes found in the cases that have hitherto been reported indicate that it is impossible to localize the lesion that gives rise to this condition. Berg tabulates eighteen cases that he has obtained from the literature, including one of his own. (The paper is still unfinished.)

3. *Myelinated Fibers in Cortex*.—Brückner continues his article upon the medullary fibers in the cortex of an eighteen-year-old Suaheli. He gives elaborate tables showing the results of his painstaking measurements, and a wealth of anatomical details that unfortunately cannot be reproduced in an abstract. He compares the results with those obtained in other human brains, particularly with that of a German child. Curiously enough the former in some respects was equally as well developed. In other respects also the brain resembled the infantile German type, particularly in the superior development of the right hemisphere.

4. *Pathology of Peroneus*.—Daus calls attention to the frequency with which the peroneal nerve is paralyzed. He classifies these as follows: (1) Congenital; (2) acquired during extrauterine existence. These may be: A. Mechanical Causes.—I. Involving the central organs; (1) external injuries either to the head or back; (2) internal mechanical injuries: brain hemorrhage, brain tumors, or lesions or tumors of the cord. II. Affecting the peripheral and peroneal tract; (1) external cutaneous injuries. (a) stab, blow, or gunshot wounds; (2) burning; (2) injuries as a result of fractures or luxations; (3) rupture of the nerve, (a) simple over-stretching, (b) over-stretching as a result of tumors; (4) pressure, (a) pressure during sleep, (b) pressure during narcosis, (c) ligation, either external, as from a bandage, or internal as the result of scars or callus, (d) compression, (e) in deformities of the knee, (f) paralysis during birth either of the mother or child, (g) tumors; (5) miscellaneous, (a) paralysis as a result of overexertion, (b) the hysterical paralysis of Charcot, (c) paralysis due to fright, (d) the paradoxical contraction of Westphal, (e) paralysis due to habit of Erb.

B. As a result of toxic causes.—I. Directly toxic; (1) chemical: alcohol, lead, arsenic, mercury; II. parasitic, (a) acute infectious disease: diphtheria, pertussis, exanthemata erysipelas, typhoid fever, pneumonia, influenza, gonorrhea, syphilis, beriberi, and malaria; (b) chronic infectious diseases: tuberculosis and leprosy; III. indirectly toxic: Chemical: Diabetes, gout, rheumatism, skin diseases, and ascending neuritis.

C. As a symptom or complication of organic disease of the central

nervous system.—I. In tabes, in tabo-paresis, in dementia paralytica, multiple sclerosis, antero-polio-myelitis, and the peroneal type of progressive muscular atrophy.

D. In old age.

E. Of unknown cause.

Daus quotes articles from the enormous literature on this subject, illustrating each of these forms. (The paper is still unfinished.)

(Vol. 13, 1903, No. 6, June.)

1. The Capacity of the Skull (The Cranial Cavity) in Nursing Infants and Older Children. H. PFISTER.
2. Disturbances in Sensory Perception in Cases of Mental Disease. W. v. BECHTEREW.
3. The Aphasic Symptom Complex. E. STORCH.
4. Contribution to the Knowledge of Transcortical Aphasia. M. BERG.

1. *Skull Capacity*.—Pfister has determined the capacity of the cranium by means of water, according to his own method, in 77 male infants, and 77 female infants, ranging in age from thirteen days to eight years. The variability in the size of the cranium in both sexes is considerable. In the second year there may be a difference of as much as 200 cm., although the average is only 948 cm. in males and 907 cm. in females. The average result for a certain period is as follows:

- I. To the end of the first month: Male, 415 cm.; female, 414 cm.
- II. To the end of the third month: Male, 503 cm.; female, 574 cm.
- III. To the end of the sixth month: Male, 651 cm.; female, 577 cm.
- IV. To the end of the tenth month: Male, 769 cm.; female, 684 cm.
- V. To the end of the fifteenth month: Male, 899 cm.; female, 883 cm.
- VI. To the end of the twenty-first month: Male, 1,030 cm.; female, 895.

As a result of his studies he reaches the following conclusions: First, the cubic capacity of an infantile skull less about 6.5 per cent, gives approximately the capacity of the cranium in the living individual. Second, the capacity of the cranium is larger at all ages in males than in females. The difference is less at birth and increases with age. Third, before the ninth month of life is completed about one-third of the total increase in the capacity of the cranium over that at birth, has occurred. Fourth, at the end of 2.5 years about two-thirds of the total increase in the capacity of the cranium has occurred. It is not known when the head ceases to enlarge. The individual variations in size are very considerable.

2. *Sensory Perception in Mental Disease*.—Von Bechterew reports some curious cases in which patients had strange disturbances of the psychical sense. A girl of twenty years, although touch, pain and temperature senses were not distinctly abnormal, had a hallucination that her head, arms, legs and even the body were missing. The condition appears to be one of purely psychical anesthesia. In another instance, an army officer had such a hyperesthesia for the word "blood" that when it was spoken, he was sometimes thrown into convulsive states. In still another case a patient had hallucinations of hearing associated with certain sensory disturbances.

3. *Aphasia*.—Storch concludes his article upon the aphasic symptom complex. He discusses perception of letters, of words, and particularly the relations of these perceptions to the various forms of aphasia. He uses the term *glosso-psyche* to indicate the entire apparatus used in the formulation of letters and the spelling of words. He devotes considerable time to the discussion of spelling, and finally concludes with a classification of the various forms of aphasia.

A.—Those without essential participation of the stereopsychic region:—intelligence and tact; (1) The *proglossopsychical* aphasias. The lesion lies peripherally to the *glossopsychical* fields (the subcortical forms, (a) subcortical sensory aphasia, or pure word-deafness: the lesion being in the

left temperosphenoidal lobe; the glossopsychic region is slightly or not at all injured; the invalid can read and write; (b) subcortical motor aphasia: aphemia and pure word-deafness: the lesion being in Broca's region. The glossopsychic region is almost intact; the patient understands speech, can read and write, but cannot speak. (II) Glossopsychic aphasias, (a) cortical sensory aphasia, the lesion having separated the glossopsychic region from the peripheral organs of hearing, and also injured the glossones. In addition to word-deafness there may be paraphasic manifestations, and disturbances of reading and writing; (b) cortical motor aphasia: the lesion not only separates the glossopsychic region from the phonetic nuclei in the medulla, but also has injured the latter. The understanding of speech and the ability to read and write are involved according to the extent of the lesion; (c) pure glossopsychic aphasia: in which only the elements of glossopsychic region are involved: therefore the understanding of speech, spontaneous speech, repetition and reading and writing are injured; (d) total aphasia in which the whole glossopsychic region is destroyed.

B.—Forms of aphasia with involvement of the stereopsychic region; (1) Pure stereopsychic forms, that is, certain varieties of transcortical motor and sensory aphasia, pure word-blindness, various form of agraphia and psychoses; (2) mixed stereoglossopsychic aphasias.

4. *Transcortical Aphasia*.—Berg continues his article with a long description of his own case. A man for forty-nine years had shown symptoms of mental impairment for several months. The essential features of the case were that voluntary speech was preserved as far as the formation of words was concerned, but the language was completely paraphasic and incoherent. The understanding for words and simple sentences was preserved, but more complicated sentences produced hopeless confusion. Voluntary writing showed pronounced paraphasia; the understanding of writing for words and simple sentences was preserved. The patient could repeat, could read, could copy, and could even write from dictation. The repetition of simple words was done intelligently. If more complicated sentences were given the paraphasic phenomenon sometimes appeared. In reading, copying and in writing from dictation the patient understood nothing. The echolala was typical. The patient was able to identify objects by sight and touch. Intelligence, memory and observation were all diminished. The case, therefore, represents a form of transcortical aphasia, partly motor and partly sensory in character, and the lesion is probably a diffuse lesion in the brain.

JOSEPH SAILER (Philadelphia).

ARCHIV FUER PSYCHIATRIE UND NERVENKRANKHEITEN

(37 Band, 1903, I Heft.)

1. Concerning Korsakow's Symptom-Complex. E. MEYER and J. RAECKE.
2. The Pathogenesis of Amyotrophic Lateral Sclerosis. H. HAENEL.
3. A Contribution to the Pathology of Huntington's Chorea. E. STIER.
4. A Contribution to the Pathology of Chorea Minor. C. HUDOVERING.
5. The Simple Demented Form of Dementia Praecox. O. DIEM.
6. The Value of the X-Rays in the Treatment of Diseases of the Spinal Cord. E. VON LEYDEN and E. GRUNMACH.
7. Further Contributions to the Pathology of Sensory Aphasia. A. PICK.
8. The Occupation Neuroses of Telegraphers. E. CRONBACH.
9. Notes on the Criticism of Dr. M. Fekch on Two Cases of Cerebellar Palsy in Children.

1. *Korsakow's Symptom-Complex*.—An investigation of the question whether multiple neuritis may be followed by definite mental symptoms. The history of eight cases is given in detail. One improved greatly so that he was practically cured; one was improved; two remained unimproved and four died. Of the four cases that went to autopsy one showed fresh degen-

eration in the crossed pyramidal tracts, another had minute hemorrhages into the gray cerebral substance, a third case showed an old apoplectic scar in the right thalamus and in the pons and the fourth had a large sarcoma in the right frontal lobe. The authors conclude that their cases show positively that Korsakow's symptom-complex is not a disease *sui generis*, also decidedly not an alcoholic psychosis, but is present also in those diseases in which irreparable or severe destruction occurs in the central nervous system.

2. *Amyotrophic Lateral Sclerosis*.—The patient was a woman forty-five years old, with good heredity, previously in good health, married, with four children. Alcoholism and syphilis were excluded. Two years previously it was noticed that the speech of the patient was awkward, also there began at that time a slowly increasing weakness in the right side, first in the arm, then in the leg. Speech was later suddenly lost although at the time of examination it was slow and indistinct, but intelligent, without paraphasia. The clinical diagnosis was bulbar paralysis and amyotrophic lateral sclerosis. Shortly before death atrophy of the small muscles of the hand, particularly on the right, was distinct. Babinski was marked on both sides. Patient became uncleanly in her habits. At necropsy the kidneys were found small, granular and with shrunken capsules; the heart was hypertrophied and dilated. A microscopical examination was made of the cerebrum, spinal cord and muscles of the tongue with Nissl, Weigert, Van Gieson and carmine stains. No new facts are added. The author inclines to the belief of a vascular toxemia in his case. A satisfactory bibliography and an excellent photograph are appended.

3. *Huntingdon's Chorea*.—One case is reported in which Nissl's thionin stain was used. Comparing his findings with those of other authors he makes the deduction that Huntingdon's chorea is always dependent upon an inherited asymmetry of the cerebral cortex. The asymmetry can frequently be seen by the unaided eye. The disease begins in middle life with proliferation of the neuroglia in the motor centers. This proliferation may be either diffuse or circumscribed and is most marked in the second and third cortical layers; that is, the layers of the small and medium-sized pyramidal cells. At the same time the blood vessels become diseased, showing outwandering of the lymphoid cells into the perivascular spaces and occasionally hemorrhage. Almost always the small and medium-sized ganglion cells become degenerated throughout, while the large ganglion cells in the innermost layer remain intact.

4. *Chorea Minor*.—A case in which bacteriological examination of the blood and a part of the cord gave negative results. Interesting findings were inflammatory changes in the central nervous system, particularly marked in the motor pathways in the pons and medulla; rounded bodies, presumably colloid, which lay in the neighborhood of the blood vessels and also free in the brain substance; light grade of ependymitis and leptomeningitis and a disruption of the cell nuclei in the cortex particularly in the pyramidal cells of Ammon's horn. (Four beautiful plates show these conditions clearly.)

5. *Dementia Praecox*.—Nineteen carefully recorded cases are added to the literature. The author makes a strong plea for the retention of the name to express a disease entity which when carefully studied shows a well-defined clinical picture.

6. *X-Rays in Cord Disorders*.—An inspiring article on the value of the Roentgen Rays in the diagnosis and treatment of affections of the spinal column with illustrative cases. Thirty patients in all were examined; in twelve the diagnosis was simply confirmatory, but in the other eighteen the diagnosis was first positively made or at least further amplified by the rays. In ten of the eighteen cases primary disease of the bony column was followed by paraplegia. These ten cases comprised compression of the dorsal medulla by tuberculous bony deposits; compression of the dorsal medulla

by kyphoscoliosis, two cases; two new growths, one carcinoma, the other sarcoma; two cases of cervical myelitis from fracture, and one case of bulbar paralysis, probably due to dislocation of the third and fourth cervical vertebrae. In the eight following cases the previous diagnosis was verified and much relief afforded by suitable treatment. One was dorsal myelitis, one, syphilitic pachymeningitis and myelitis, and one, lumbar myelomeningitis, and two cases of tabes dorsalis. In all of these distinct osteoporosis was diagnosed by the rays. In another case diagnosed as tabes an osteoarthropathy of the spinal column was further diagnosed, and in still another case of tabes with arthropathies of both knees the diagnosis of osteoarthropathy of both knees was made and an osteoporosis of the spinal column was shown.

7. *Sensory Aphasia*.—Will be abstracted when completed.

8. *Telegrapher's Neuroses*.—But sixteen previously reported cases could be found. The author first reviews the literature and then gives the history of his own case, which showed nothing of particular note. In eleven of the total cases it is interesting to observe that the affection began in the spring months. A neuropathic tendency is shown by nervous weakness in the immediate family in six of the cases, by apoplexy in three cases, by gout in two cases and by stomach trouble with convulsions in one case. Complications with other diseases was noted in nine cases. The treatment promises little. The therapeutic measures advised are great, change of scene, baths, cold water cure, faradism, galvanism, massage, gymnastics, change of occupation or of the method of work. Of these measures massage gives the best results, while change of occupation did not give the benefit one would expect. The use of the Morse instrument intermittently with the Hughs instrument is advised as a prophylactic measure.

9. *Cerebellar Palsy*.—This is a caustic reply of Dr. Sommer to the criticism of Dr. Felch. (36 Bd. 3 Hft, S 895 et al.)

F. WITMER (New York).

AMERICAN JOURNAL OF INSANITY

(Vol. 59, 1903, No. 4, April.)

1. A Contribution to the Study of the Blood in Manic-Depressive Insanity. J. S. FISHER.
2. The Hallucinatory Delirium of Acute Alcoholism. C. S. WALKER.
3. The Progress of Psychiatry in 1902. A. R. URQUHART.
4. Puerperal Insanity. R. JONES.
5. The Care of the Insane. C. G. WAGNER.
6. Tent Life for the Insane. O. J. WILSEY.
7. A Graded and Systematized Plan of Outdoor Exercise for the Demented Insane. C. L. CARLISLE.
8. The Final Chapter in the History of an Extensive Injury to the Head. H. P. FROST.
9. Therapeutic Notes. R. DEWEY.

1. *Blood in Manic-Depressive Insanity*.—Owing to no account being taken of data that may have an influence on the condition of the blood, the author of this paper made his observations at the same time of the date under uniform relations as to food, baths, bodily temperature, etc. His blood counts were confined to five cases of manic-depressive insanity with care that the technic be identical. The first case was a female of nineteen with the maniacal form. On admission had hallucinations of hearing, consciousness clouded, disoriented as to time and place, memory defective, rambling in her conversation; no well defined delusions, great motor excitement with tendency to impulsive acts. No disease insight. Temperature always normal. Fifteen counts were made. The next case was a male of

thirty with the maniacal form, consciousness clouded, flight of ideas, etc., yet oriented, memory unimpaired. Six counts were made. The third case was a mixed form in a female of forty-seven. Consciousness somewhat clouded, some retardation of thought and action; no delusions, no interest in her environment. Twenty-two counts were made. Case four was the maniacal form in a female of thirty-three. She had hallucinations of hearing, consciousness clear, oriented as to time, place and persons, no memory defect, judgment biased by delusions, no disease insight, had little interest in her surroundings. Forty-five blood counts made. The last case was of the maniacal form in a woman of fifty-seven. No hallucinations, consciousness clouded, mistook those about, completely disoriented, psychomotor activity marked. Only four counts made. The results of his examinations he summarizes in a table and draws the following conclusions from his observations: "1. There are no pathogenic blood changes in cases of maniacal phases of manic-depressive insanity. 2. Anemia is not a causative factor nor always an accompaniment of this psychosis. 3. The hemoglobin and red cells are frequently, if not always, increased during an attack of excitement. 4. A leucocytosis is an almost constant accompaniment and, I believe, a result of psychomotor activity. 5. That an increase in weight accompanies mental improvement."

2. *Hallucinatory Delirium of Acute Alcoholism*.—The statement of an acute alcoholic detailing the characteristic hallucinations of this disease with the delusions arising therefrom.

3. *The Progress of Psychiatry in 1902*.—Urkuhart mentions the paper of Dr. Warnock on Pellagrous Insanity and two papers by Dr. Mott, pathologist of the London County Council. The first deals with Stimulus in Repair and Decay of the Nervous System, setting forth the danger to neuropathics in the use of alcohol. He claims that stress is an important factor in the etiology of tabes. The second paper treats of syphilis as a cause of insanity. He states 70 to 80 per cent of paretics are found to have had syphilis and accepts the thesis: "No syphilis, no general paralysis." Dr. F. G. Crookshank's paper on "The Frequency, Causation, Prevention and Treatment of Phthisis Pulmonalis in Asylums for the Insane, as well as Dr. Eric France's paper on "The Necessity for Isolating the Phthisical Insane," has led to greater interest in this important subject, and the means for prevention urged are: early diagnosis, isolation, limitation of size of asylums, checking of overcrowding, improved ventilation, attention to dietary and sanitation in detail. Asylum Dysentery has been given careful study and is found not to essentially differ from ordinary dysentery. Night nursing has been given considerable study, with better care of patients at night. Marked advancement has been made in the matter of training nurses. The London County Council has established a Laboratory of Research at Claybury Asylum, where much attention has been given to clinical problems. Toxemia has been studied carefully and Dr. Ford Robertson claims all forms of insanity in normal persons is of toxic origin. An effort has been made, with some success, to treat incipient mental disorders in general hospitals. Some study has been given to asylum dietaries. Dr. Mercier's two books, a small Text-book on Insanity, the other a systematic treatise on Normal and Morbid Psychology, are notable books. The paper devotes itself solely to progress in England.

4. *Puerperal Insanity*.—The percentage of insanity due to pregnancy, parturition, the puerperal state and lactation, according to the report of the English Lunacy Commission, for five years, amounts to 6.4 in the private class and 8.1 in the poorer classes. At Claybury the statistics closely correspond to the above. Of 3500 admissions 259, or 7.4 per cent, suffered with insanity from the above specified causes, and are divided as follows: 56 from pregnancy or 21.62 per cent, 120 occurred during the puerperal state, or 46.33 per cent, and 83 were associated with lactation, 32.43 per

cent. The type of disease with pregnancy and lactation has nothing distinctive, while that of the puerperal state is marked by intense delirium with wildness and hallucinatory delusions, with permanent religious and erotic traits. Some cases present some mental disturbance to develop to insanity in the puerperal period, others again present mental symptoms during the puerperal state to be markedly alienated during lactation. Only 12 per cent of the cases were unmarried, yet of the insanity of pregnancy 25 per cent were unmarried, 8 per cent of the puerperal cases and only 3 per cent of the lactation cases. In the insanity of pregnancy it is mostly of the depressed type, while in the puerperal cases a maniacal type prevailed, and of the lactation cases the majority were of the depressed form. In 40 per cent of the cases of puerperal insanity symptoms appeared within the first two weeks and more than a third of these during the first week. Almost without exception the early symptom was loss of sleep followed by restlessness, distrust and suspicion, irritability followed by delirious excitement. Hallucinations of hearing were six times more frequent in these cases than of any other of the senses. Disorientation as to time and surroundings is common, as well as that she fails to recognize her own identity. The confusional state is followed by uncontrollable and restless violence, attended by profound physical exhaustion. The patient is often antagonistic to her husband, has erotic delusions, is immodest in conduct and language. Marked sexual excitement often exists. Suicidal thoughts are most common in the lactation cases occurring in nearly half, while in insanity of pregnancy it was noted in 41 per cent, in the post-*puerperal* in 21 per cent. The tendency to injure the child more common to the lactation cases. A previous record of hysteria is found in quite a percentage of these cases, but it is hardly sufficient to advise hysterical girls not to marry. Brown hair and gray eyes predominate. The age of greatest incidence was between twenty-five and twenty-nine for the insanity of pregnancy, and that of the *puerperal* period, while in the lactation cases from thirty to thirty-four. Heredity was present in 50 per cent and most so in the *puerperal* and pregnancy cases. This is considered the most favorable form of insanity, yet is very hazardous to the offspring. Insanity in early pregnancy is favorable and often recovers before confinement, while that of later gestation is apt to continue in an exaggerated degree and may become chronic. Most of the *puerperal* cases recover quickly, although permanent dementia may result. Cases of sudden onset show a slightly greater tendency to recover than those of gradual. Albuminuria, which is frequent, renders the prognosis grave. Gradual onset occurred in the cases becoming chronic and those dying. The death rate is the highest in the insanity of pregnancy and lowest in the *puerperal* cases. The pathology of these forms of insanity is not definitely established; some hold that various products of the changes incident to the processes of pregnancy and the *puerperal* state are the cause, in order words toxic, while in the lactational insanity the exhaustion often incident to this process may be the chief cause. The treatment must naturally depend on the fundamental condition. The induction of abortion in insanity of pregnancy has not proven beneficial. The patients need constant watching owing to their sudden outbursts of violence and suicidal ideas. Commitment to an asylum is generally best, unless the patient is so situated financially that she can have all the advantages of an institution provided in her own home. *Puerperal* cases should be treated at home if possible. The essential treatment is "compulsory superalimentation." Sulphonal is useful to induce sleep in motor excitement, paraldehyde is satisfactory, but not so good as a combination of chloral and bromide. A dull, listless condition may occur from which the patient is to be roused by a change in surroundings. Complications are to be treated according to their indications.

5. *The Care of the Insane*.—The last quarter of the nineteenth century

has witnessed wonderful strides in the care of the insane throughout the world. Until very recently the bars, locks, crib-beds and massive furniture were appalling, to say nothing of the devices for mechanical restraint. Of course this innovation in the treatment of a class considered so dangerous met with violent opposition. Exercise was confined to the "bull-yard," while now many patients are permitted almost unbounded liberty. Further occupation has been provided for most of the patients, instead of being compelled to spend their days on the ward benches, they are now helping in the shops, kitchen and farm work. Instead of the wards being bare of all ornamentation and hence cheerless and depressing, they are now comfortably and attractively furnished. The treatment of the insane today consists of "pleasant and sanitary surroundings, good nursing, proper medical attendance, suitable diet, entertainment and congenial occupation." One of the crying needs still is suitable buildings and appliances for the treatment of the acute cases, possibly recoverable under proper care, where association with chronic cases would be avoided, the appliances, both medical and surgical, provided for correcting and relieving physical ailments to which the mental disorder is often due. This would be a saving to the State in the long run by making the patient again productive, instead of possibly dependent for the balance of his life.

6. *Tent Life for the Insane.*—Dr. Wilsey cites in detail the benefit to a case treated in a tent nearly twenty years ago, and with marked and lasting improvement and recovery, and believes "the complete change in the whole existence of the patient was the important curative agents," and he warns us not to let means of diversion become monotonous, and those physicians who are the most versatile in providing for the patients' mental diversion will be the most successful.

7. *A Graded and Systematic Plan of Outdoor Exercise for the Demented Insane.*—The physical health demands the first attention in the amelioration of advanced dementia. A sclerotic state of the vascular system is largely responsible for the atrophic condition of the alimentary tract, resulting in more or less chronic states, which cause the defective metabolism. Consequently the dietary must be suited to this condition and therefore quality is more important than the quantity of food, and it must contain the largest possible amount of tissue-building material with the smallest possible amount of detritus. Digestive ferments and other medication may be employed to improve the patient's digestion. An improvement in the mental condition soon follows the betterment of the digestive process: the patient becomes more attentive, more tidy in his habits and more intelligent in his actions. The next essential is an abundance of exercise in the open air, and for this purpose the patient should begin with the simplest possible, one requiring little or no mental effort, and in time the patient will be found capable of performing more complicated work and even making himself useful in the shop. The games and occupation should be adapted as much as possible to the patient's individual liking. The faithful systematization and gradation of the amount and sort of out-door exercise has reduced the number of filthy cases to less than one-fifth of one per cent of the entire population of the hospital. The etiology and type of the disease must of course influence the possibility of either permanent or partial improvement. The most marked benefit has occurred in secondary dementia, particularly that following melancholia, while that following mania is less susceptible to instruction and suggestion. Dementia from organic brain disease is to be the least benefited. Great benefit is to be derived from this method of treatment in psychopathically inferior individuals and the allied forms of adolescent insanity, including hebephrenia and dementia precox. The results already attained warrant its further development and use.

8. *Extensive Injury to the Head.*—This case has been frequently re-

ported at various times from that of the receipt of the injury in 1857 until the patient's death within the past year. The first account embraces the details of the accident, which consisted of the tearing away of nearly the whole of the right side of the calvarium from the superciliary ridge to the occipital bone. At the time the patient suffered only slightly from shock, but no signs of cerebral concussion, faintness or nausea. The wound soon healed with no further symptoms than a dull headache. This report appeared in the *Buffalo Medical and Surgical Journal* for October, 1873, with the statement that the patient was in vigorous health at the time and only experienced a sense of fulness in the head when stooping or exerting himself. The next report of the case was by Gray, published in the *American Journal of Insanity*, April, 1876. This was followed by Dr. Bergtold's article in the *Medical Press of Western New York*, in 1888, written from his observation of the patient in the Buffalo General Hospital the year previous. In 1883 a slight unsteadiness of gait appeared which in 1888 had become a genuine paralysis, affecting the left extremities and right side of the face. Memory intact, no aphasia or headache, yet occasional attacks of dyspnea of asthmatic character; no cystic paralysis, yet some loss of control of the rectal sphincter. Sleep and appetite good. Deficiency of bone on right side of head with a depression of 5 x 6 inches. Special senses normal, speech slow and labored from paralysis. Mastication and swallowing interfered with, tongue protruded to left, lower lip pendulous. Slight atrophy of trunk and extremities, left arm nearly powerless, spastic pronation of hand and flexion of fingers, spastic contraction of left leg which was dragged in walking. Cutaneous sensibility intact; muscles respond to galvanic and faradic currents. No delusions or hallucinations, but patient irritable and emotional. Dr. Bergtold drew the following conclusions: "There probably began immediately subsequent to the injury an atrophy of the cerebral tissues situated beneath the site of the right parietal bone; this atrophy included the motor centers for the left arm and leg and the cortical centers for the facial and hypoglossal nerves; the atrophy, being slow, the remaining right cortex took on vicariously the duties of these atrophying centers; there gradually occurred (within the past five years) degenerative changes in these right compensating parts and further compensation did not take place in the left hemisphere." Thirteen years later he was admitted to the Buffalo State Hospital, being quite helpless and his mental faculties more affected. He was childish and irritable with paroxysms of anger, when we would try to strike. He could use his right arm, but both legs were firmly flexed at the knee and the right leg could only be moved feebly. Left arm spastic and joints contracted. Knee-jerks exaggerated, especially on the right. No paralysis of right side of face, tongue and lips paretic, eyelids drooping, tongue tremulous and atrophied, but protruded in the middle line without deviation; swallowing interfered with, speech almost unintelligible, drooling of saliva constant. Marked external strabismus, left eye noticeably protruding. Pupils unequal, sluggish in reaction to light and accommodation, vision imperfect, hearing about normal. Sensation apparently intact. Memory not seriously impaired, appreciative and tidy. No convulsions or attacks of unconsciousness. Died two years after admission. Autopsy showed a large deficiency of bone of the skull, where the scalp was adherent to the dura, which was thickened. Whole right hemisphere flattened and atrophied, but no evidence of injury or loss of brain tissue. Convolutions noticeably smaller than on left, of a shrunken, atrophied appearance, sulci gapping. No atheroma of the blood vessels. After hardening the brain was sectioned according to Hamilton's method, when a marked difference in the size of the hemispheres is shown. The most striking feature was a considerable area of degeneration of the subcortical white matter with numerous cavities just beneath the cortex and the hollowing out of certain convolutions. Slight softening of white

matter through whole right hemisphere, yet no breaking down. Cavities confined to individual convolutions and not connected or noticeable externally except by slight depression. No other marked changes. The examination after staining showed a diffuse fibroid thickening and degeneration of the walls of the blood vessels in both hemispheres, but most marked in the right, the lumen being occasionally occluded. The vessels here showed a decided infiltration of round cells. The vessels on the right fuller than on the left. But few of the large pyramidal cells left and these shrunken and degenerated. A general increase of the neuroglia elements throughout the tissue. On the left side there is a slight degeneration of the tangential fibers in the cortex with projection fibers preserved; on the right tangential fibers absent at the top of the convolutions, yet seen at the sides. Degeneration of both motor and sensory tracts in the medulla and cord, most marked in those from the right hemisphere. These conditions in the cord explain the exaggerated reflexes, ataxia, spastic rigidity and gradual progress of the paralysis. The increased atmospheric pressure on the brain had diminished the capacity of the lymph canals and blood vessels, thus inducing the general atrophy from lack of nutrition, and this being progressive explains the character of the paralysis. The paper is accompanied by several photographs of the patient and the brain sections.

9. *Therapeutic Notes.*—*Cascara.*—Dr. Dewey extols the preparation known as "Kasagra," it being palatable and efficient. He advises that it be given in a dram before breakfast followed by a glass of hot water, and one or two drams followed by water at bedtime. It has proven effectual in the great majority of cases, even in those where constipation is a habit. Serum treatment of morphine intoxication. The researches of L. Hirschlaff are reported, who prepared a serum from rabbits by giving them morphine in increasing doses, which he found efficacious in opium poisoning and morphine addiction. Use of iron of animal origin. Hemoglobin may be given successfully in anemia and chlorosis. Mental disturbances due to hypnotism. Prof. Mendel's report is: "Careful distinction must be made between the cure of an affection and the alleviation of its symptoms by hypnotism. No physician claims that it is possible to cure with it an organic affection susceptible of demonstration with our modern diagnostic measures. But hypnotic or waking suggestion is undoubtedly capable of banishing, usually temporarily, but sometimes permanently, the most widely diverse symptoms of the affection, but without curing it. Success is dependent in this case, as in all cases of suggestion, on the greater or less skill of the suggestor, his personality, on external circumstances and on the degree of receptivity of suggestion by the patient. A number of cases are known, in which mental disturbances developed in predisposed individuals under hypnotization. Some of these cases occurred with charlatans, but in others the hypnosis had been conducted by physicians." Arteriosclerosis. The causes are cited and the treatment outlined, of which intestinal antiseptics is an important factor. The diet advisable is given in detail.

W. A. McCORN (Patterson).

MISCELLANY

TWO CASES OF SARCOMA OF THE SPINAL CORD. Senator (Prakt. Vrach., No. 24, 1903).

Tumors of the cord, until recently varieties inaccessible to treatment, have of late excited considerable interest. The author presents two highly instructive cases. In one case, that of a woman, sixty-nine years of age, there was a history of ten months' duration, that began with a sensation of formication in the toes and pains in the feet; to this was later added inability to flex the knees, burning and pains in the soles, which spread upward to the hip joint. During the last six months patient lost all power of locomotion, suffered from convulsive twitchings in the lower ex-

tremity, at times so pronounced and painful that the knees were forcibly flexed over the chest. There was also retention of urine. Patient lies recumbent on the back and is unable to move the lower extremities. Passive movements also hardly possible. Knee reflexes very much exaggerated. Babinski's sign present. Percussion of the muscles calls out painful twitchings. Stroking of the soles or legs is attended with clonic contractions of the lower extremities and of the abdominal muscles. Abdominal reflexes absent. Sensation lowered in the lower extremities. This condition grew gradually worse until the sensation of pain and temperature entirely disappeared, while that of touch disappeared over a still greater area, extending upward. Late in the disease there occurred curvature of the spine at the third lumbar vertebra. In the course of the disease the body of the patient grew, as it were, shorter, the chest approached the pelvis. The abdominal muscles were almost in a continual state of contraction. Dyspnea and attacks of tightness in the chest, Cheyne-Stoke's respiration and marked slowness of the breath (eight per minute) supervened. Lumbar puncture during the early period of the disease brought forth 10 to 12 cc. of a limp liquid, with but little albumin. After the second puncture 5 cc. of one-half per cent solution of cocaine was introduced into the subdural space with remarkable improvement to the contractures and pains, which lasted for three hours. Of the drugs employed, scopolamine (hyoscyamine) seems to have given the best results. The autopsy revealed a tumor of the dura mater, which was microscopically seen to be a sarcoma. The second patient was admitted to the hospital with pains in the back, inability to move the legs, and complaining of tormenting thirst. The trouble began seven months before with pains in the back, between the scapulæ, which would at times become so severe as to render her unable to work. Some four weeks before admission, the soles began to swell; this was followed by swelling of the abdomen, while two weeks ago patient became unable to move the left leg. There is no retention of either urine or feces. Patient recumbent on the back, indifferent, consciousness slightly impaired. Knee reflexes exaggerated, Babinski's sign present, ankle clonus indistinct. It is rather difficult to pick up the patient, as the spinal column is quite stiff. Patient complains especially of pains in the legs. Autopsy (patient died on the third day of admission to the hospital) gave the following: The heart slightly hypertrophied, lungs somewhat adherent, the thyroid gland hypertrophied, full of cysts; the bladder distended, its mucous membrane covered with necrotic spots; ovaries thickened. The vessels of the brain markedly calcified; the spinal column much congested; in the region of the third and fourth thoracic vertebræ, to the left, there is seen a hard swelling, which is microscopically a fibro-sarcoma. The interesting feature of this case is the very rapid course of the disease. The first stage, what the author calls the stage of neuralgia, was quite short, while the stage of paraplegia, that usually occupies months and years, was hardly extended over two weeks.

ROVINSKY (New York).

CASE OF TUMOR OF THE SPINAL CORD, WITH NECROPSY. Sinclair Gillis and Flashman (Australasian Medical Gazette, Nov. 20, 1902).

Diagnosis based on history of numbness in left hand (seven months' duration), followed by weakness in left hand and arm, and later by weakness in the right arm and in legs. Marked analgesia, anesthesia slight, except in left hand; anesthetic and analgesic areas being dissociated, thus, apparently indicating lesion of central gray matter in upper cervical region of cord. The existence of neuralgia, to a certain extent, in the left occipital region, tactile sense but slightly involved, indicated, possibly, syringomyelia. Subsequently there was great spread of analgesia (slight involvement of tactile sense), wasting of muscles of right palm, rigidity and weakness of

right arm, followed by same condition in the left, spastic paralysis of lower extremities, involvement of sphincters, absence of pain, and, finally, death by bulbar paralysis. Postmortem: Brain and meninges normal. Attached to the spinal dura mater, on the right side, and a little posteriorly, about a quarter of an inch below the foramen magnum, was a growth the size of a shelled walnut. It was irregularly bossed, and attached to the dura by a narrow pedicle, and lay free in the subarachnoid space. It protruded through the foramen magnum one half inch, lying behind and to the right of the cord, which it compressed in the region of the first and second cervical segments. Cord here somewhat softened. A horizontal section close below the foramen magnum bisected the growth, leaving the lower fragment attached a quarter of an inch below to the dura mater, and the upper by a fine filament, to the medulla. Cord not invaded by growth, and, beyond softening in region of growth, microscopically normal, section of growth showed it to be a sarcoma, round celled variety. Nerve cells, all regions of spinal cord normal (Nissl method). Few scattered degenerated fibers in the ventrolateral white columns, especially ventral portion (Marchi method). Nearly whole medulla exhibited extensive fiber degeneration, probably due to pressure. Both inferior cerebellar peduncles showed well marked degeneration, affecting one-third of fibers, due to pressure on direct cerebellar tracts and external arcuate fibers. Pyramidal tracts slightly involved. Degeneration up into pons, limited to region of mesial fillet.

J. E. CLARK, New York.

ARTERIO-SCLEROSIS AS A CAUSE OF NERVOUS DISEASE. M. Allen Starr
(Medical Record, July 4, 1903).

A careful study of two hundred consecutive cases of apoplexy in private practice has shown that in 80 per cent of these cases there have been distinct prodromata of the apoplectic attack. There were various symptoms which have usually been regarded as neurasthenic, pointing to a disturbance of function in the brain, chiefly in the cortical activity. Dulness and hebetude, difficulty of clear thinking or of remembering events of recent occurrence, or a general sense of perplexity. Others complain of a temporary sensation of numbness in one limb or one side of the body, or pains, or visual or auditory symptoms. Such symptoms are to be traced to malnutrition of the neurones, resulting from arterial disease. The writer advises thorough investigation of the condition of the blood vessels, especially the heart, and tension of the arteries, and when the condition of arterial sclerosis is recognized, appropriate treatment will make much of the so-called neurasthenia of middle age disappear.

The majority of the diseases of the spinal cord are equally traceable to disease in the vessel wall. Anterior poliomyelitis, bulbar paralysis, and ophthalmoplegia, when not distinctly infectious, are due to rupture or thrombosis of some branch of the anterior spinal artery supplying the anterior gray horns of the spinal cord. Myelitis, whether disseminated or transverse, if not due to infection, is due to a rupture of a blood vessel which has suffered from disease. Spastic paralysis, commonly known as Erb's syphilitic paraplegia, is now recognized as due to malnutrition of the dorsal region of the spinal cord, with consequent descending degeneration of the lateral columns. The cause of the malnutrition is syphilitic endarteritis obliterating the spinal blood vessels. Senile paraplegia, in which a gradually advancing weakness culminates in a slowly progressive paralysis of the legs, with imperfect control of the rectum, is due to obliteration of the blood vessels, to the thickening of their walls, and to a consequent malnutrition of their parts. Combined sclerosis is due to the same cause. Disease of the peripheral nerves is occasionally due to endarteritis of the small vessels. The various forms of neurasthenia and neuralgia are fre-

quently due to the same cause. Treatment by iodide of potassium is frequently indicated for a time in high tension pulse. The writer believes thyroid extract is of use in some cases of temporary high tension pulse, and has noted especial results in cases of migraine, where the tension is usually high.

W. B. NOYES, New York.

MENINGITIS WITHOUT ANATOMICAL LESIONS. J. Birnbaum (Münchener medicinische Wochenschrift, July 21, 1903).

It occasionally happens that patients die very suddenly with all the symptoms of a severe meningitis and yet only a slight exudate and perhaps a moderate degree of swelling and hyperemia of the brain is found post mortem. The history of the case reported by the author is unique in that the patient was ill for fifteen weeks, and that absolutely nothing was found post mortem in the meninges. Bacteriological examination, however, revealed the presence of a *meningococcus*, and the cerebrospinal fluid withdrawn during life showed a slight coagulum. The case was looked upon as one of meningeal sepsis without anatomical lesion. The cause was probably infection from a gum-boil which had been incised with extraction of the diseased tooth, two days before the acute onset of the illness.

JELLIFFE.

PROCEEDINGS OF THE ANNUAL SESSION OF THE GERMAN ASSOCIATION FOR PSYCHIATRY, AT JENA, April 20, 21, 1903.

The following critical summaries and papers were read and discussed. A critical summary upon the employment of isolation in the treatment of cases of mental disease, by Mercklin; papers upon the anatomical cortical region, by Vogt; upon the cytohistological localization of the human cortex, by Brodmann. A second critical summary upon the pronouncement of the Prussian Minister of Justice on the 9th of October, 1902, concerning the conduct of experts in the determination of irresponsibility, was read by Thomsen. A report upon the activity of the statistical commission named in the past year, was read by Hoche and Aschaffenburg, and a paper on the contribution to the knowledge of cretinism was read by Weygandt. Wollenberg demonstrated preparations of brain tumors. Liepmann read a paper upon the course of ideas. The third critical summary upon the idea and the significance of dementia, was read by Tucsek. Papers were read upon the construction of a sanitarium for nervous patients at the public expense, in Rasenmühle, in Göttingen, by Cramer on the experimental studies with reference to the pathogenesis of acute psychosis, by Berger; upon the significance of special medication in the treatment of imbeciles, by Lacquer, and Binswanger demonstrated three cases of postsyphilitic dementia.

JOSEPH SAILER, Philadelphia.

TABES AND THE MARITAL RELATION. A. Pitrés (Journal de Médecin de Bordeaux, July 12, 1903).

The author here presents a statistical article based on a study of 240 tabetics and dealing with the influence of tabes upon fecundity and the health of the offspring in marriages in which one parent was afflicted with locomotor ataxia. The 31 celibates of this number are dismissed by the author without comment. The issue of the remaining 209 amounted to 483 children; of whom 197 were still-born or died in the early months, and 286 survived; making an average of 2.31 births to each couple, with but 1.36 living children. Carrying the analysis further, the author states that of the 209 marriages, 42, or 20 per cent, were absolutely sterile, 32, or 15 per cent, resulted in still-births or death shortly after birth; while in the remaining 135, or 60 per cent, there were living children in the relatively high proportion of 2.11 per family. The high mortality seen in the offspring of tabetics,

the author believes, is attributable to the antecedent infection rather than indirectly to tabes; and this infection is, in the majority of cases, syphilitic. He cites figures which go to show that the mortality of children born before development of tabes in either parent was 44 per cent.; while those born after the appearance of tabes showed a mortality of but 28 per cent. This finding furnishes, in his opinion, additional proof of the statement made by Régis, to the effect that the offspring of tabetics have a better chance of life and health when born in the later stages of the parents' disease than at a period nearer to the antecedent infection. As to the outcome of his investigations looking to the determination of the influence of syphilis upon the mortality of tabetics' children, the following figures are quoted: Of 77 male subjects who had undoubtedly had syphilis, 187 children were born, of whom 33 per cent were still-born or died in early infancy, and 67 per cent survived. Of 34 who denied antecedent syphilis, 81 children were born, of whom 20 per cent were still-born or died shortly after birth, and 80 per cent survived. With few exceptions, Pitrés finds that the surviving children of tabetics are uninfluenced by the parents' disease; their physical and intellectual development proceeding normally as that of other children; and of the 286 children of tabetic parents, whom he kept under observation, many were alive at twenty years, and some had attained thirty and thirty-five years at the time of writing. In none did tabes or Friedreich's disease develop, neither was there evidence of any other nervous affection. None showed signs of physical or mental degeneracy, nor was the influence of syphilis apparent in any. On the other hand, some developed diseases independent of all hereditary influence; such as tuberculosis, rheumatic cardiopathies, etc. The great majority were, however, healthy in body and mind.

JELLIFFE.

CHOREA IN PREGNANCY. Cecil Wall (*Brit. Jour. of Obstet.*, June, 1903).

It has often been observed that the movements of a choreic patient are closely akin to those normally employed in the expression of emotions. In a young child, before the highest centers have developed coordinating control, such movements are alone present; it is only by a process of education that spontaneous movements of early infancy become coordinated, and thus capable of purposive action. Rheumatism is undoubtedly, in the majority of cases, associated in some way with the chorea of childhood. So, too, during pregnancy, rheumatism plays an important part in the etiology of chorea. Buist found a personal history of rheumatism in 45 out of the 226 cases that he analyzed. The author's statistics show that at least 16 of 37 patients had previously suffered from some form of rheumatism. Twelve more had had chorea in childhood without other rheumatic manifestations. Antecedent chorea may suggest a rheumatic taint, but cannot be taken as conclusive evidence. In pregnancy, defective mental development seems to vie with rheumatism and previous chorea as one of the predisposing factors, rendering patients liable to an attack of chorea. The determining cause for chorea is not infrequently to be found in worry, for which the pregnancy is the chief cause; in case the patient is worried by the knowledge that her child is illegitimate. Fear of dystocia may be another inciting factor. Chorea in pregnancy seems to be determined by mental display, overstrain and shock. Secondly, the determining cause is only effectual when it acts upon a brain whose power of control is somewhat lowered by the pregnant state, and in addition is unstable in consequence of (1) antecedent chorea; (2) antecedent rheumatism or a similar debilitating condition; (3) a defect in development.

JELLIFFE.

Book Reviews.

A DICTIONARY OF MEDICAL SCIENCE. By ROBLEY DUNGLISON, M.D., LL.D. Twenty-third Edition. Edited by THOS. L. STEDMAN, A.M., M.D. Lea Brothers & Co., Philadelphia and New York.

Dunglison's Dictionary has always occupied the first rank among the medical dictionaries of this country, and in its successive editions it has been brought more or less up-to-date. With the present edition, the 23d, it has undergone a much more radical revision than has ever before taken place in its history, bringing it once more into the foreground as a serious and trustworthy dictionary.

Notwithstanding the immense number of new terms which have been added, the work has been kept within reasonable limits and the editor is to be congratulated on his careful work in the field of cross-reference and in his elimination of much waste matter. We can congratulate both editor and publisher alike on the successful accomplishment of this twenty-third edition.

R. BROWN (New York).

Mikroskopisch-topographischer Atlas des Menschlichen Zentralnervensystems mit beuleitendem Texte von Dr. Otto Marburg (Ehemaligen Assiatiulin au Neurologischen Institute der Weiner Universität) Mit Eineun Gleitnost von Prof. Dr. H. Obsteiner. Mit 5 Abbildungan un Texte und 30 Tafeln nach Originalin des Akademischen Malers A. Kiss. Franz Deuticke, Leipzig and Wein, 1904.

This work embraces the author's well known systematic scheme of study of the central nervous system. His experience is shown in that this work is concise, lucid and practical. The Atlas is particularly timely as clinical neurology of today demands an accurate working knowledge of nervous anatomy. American colleges are just inaugurating this work. The Atlas consists of 125 pages of descriptive text illustrated by five text figures and thirty full-page plate drawings. The latter are especially beautiful and clear. The sections are stained after Weigert-Pall and Czokor carmine. The serial and natural order in which the spinal cord, medulla pons, brain stems and brain are considered, renders it easy for the student to gain the essential and practical facts. Sections in three planes aid much in orientation of the important anatomical structures. An introduction by Prof. Obersteiner accompanies the work. We can heartily recommend this working atlas of the central nervous system to every thorough-going student of neurology.

L. PIERCE CLARK.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

ENORMOUS TUMOR OF THE POSTERO-PARIETAL REGION,
WEIGHING OVER HALF A POUND; ABSENCE OF LO-
CALIZING SYMPTOMS UNTIL LATE IN THE HIS-
TORY OF THE CASE; OPERATION; DEATH.

History of the case by Dr. Dercum.

BY F. X. DERCUM, M.D., AND W. W. KEEN, M.D.

The following case, because of the extreme difficulty of localizing the growth, of the nature of some of the symptoms present, and because of the enormous size of the tumor, is of unusual interest and importance.

H. W., age twenty-six, single, formerly civil engineer, later hotel proprietor. Examined April 23, 1902.

Family History. Mother died at the age of thirty-six with pulmonary tuberculosis. Father living and in good health. Patient has one sister, who is also living and well.

Personal History. Was healthy at birth. At the age of one year, suffered from a discharge from the right ear and subsequently lost his hearing upon this side to a very decided degree. An examination made some years later revealed a perforation of the drum. At long intervals, discharge from this ear recurred. During the past year or two the ear has again been a source of annoyance, as it has again been discharging a thick offensive fluid.

The patient had the ordinary diseases of childhood, from which he made good recoveries. He has had, since childhood, no serious illness, that is nothing that confined him to bed. He had at various times occasion to consult physicians for indi-

gestion and occasional attacks of headache. He had been educated as a civil engineer, and some years ago, while working in some mines in Kentucky, had received at various times quite severe blows on the top of his head, but not severe enough to cause loss of consciousness. At another time his legs were seriously bruised by an accident.

For a year past he has been manager of a hotel at a seaside resort, and in March, 1902, his hotel was suddenly placed in danger from fire. At this time he exerted himself very greatly in order to secure the safe exit from the hotel of his guests and other persons, and also to save their trunks and valuables. Although he had been placed under sudden and serious strain, he betrayed no special symptoms save some headache. This headache persisted, and he believed that it was in some way connected with his eyes. He thereupon consulted Dr. G. E. de Schweinitz. Dr. de Schweinitz reports that he found at this time no changes whatever in the eye-ground, nor any defect of any of the ocular muscles. About one month later, on April 23, the patient again visited Dr. de Schweinitz, and upon this second examination a double optic neuritis was observed. It was most marked in the left eye, the field of which was somewhat contracted. There was also paralysis of the right external rectus and a consequent diplopia.

On April 23 he was first examined by myself. Questioned again as to his past history, he now recalled that for a number of months past he had noticed a little uncertainty of movement in the right arm, that he noticed occasionally that he would make a mistake in the act of picking up his pen, that is, that his fingers would not grasp the pen but would go a little to either side of it. He distinctly recalled that upon one occasion when about to help himself to butter he put his fingers instead of his knife into the butter. These acts of slight loss of control of the movements of the right arm were not, however, frequent. He also noticed awkwardness of movement in his legs, in trying to go upstairs two steps at a time. He added also that he had recently had several attacks of vomiting, especially in the early morning, before he had taken any food.

Present Condition. Complains of a dull headache. Asked to localize it, he states that it is more marked over the left eye. At times the headache is diffuse over the entire forehead. He has no headache over the parietal region, over the vertex or over the occiput. At times he has a feeling of tension about the head and at times also he is dizzy. Occasionally, if he stoops to pick up something from the floor, he is obliged to get up slowly; otherwise the pain in his head grows worse. Has no ringing in

the ears. Has not vomited for several weeks past, but not infrequently feels inclined to vomit.

Physical Examination. Gait normal. Station normal. States, however, that he has a feeling of uncertainty while making the Romberg test. Stands fairly well upon either leg alone. There is no intention tremor. Tongue protruded in the median line. The tongue is slightly tremulous. There is also a little tremor and unsteadiness of the lips. The left eyelid droops slightly. The right nasolabial fold is possibly a little shallower than the left. There is no inequality of the mouth. There is marked deafness of the right ear, which is due evidently to an old otitis media. The grip of the right hand is 84; the grip of the left hand is 74. There is no change in the handwriting, though the patient himself declares that it is somewhat shaky. There is no ataxia. Both knee-jerks are minus, but are well reinforced. There is no ankle clonus. The Achilles jerk is present and apparently normal. There is no Babinski sign. There are no sensory losses, though the patient complains of a feeling of numbness upon the inner aspect of the lower portion of the right thigh and upon the inner aspect of the right calf. There is loss of stereognostic perception.

His pulse is 70 and his temperature is 98.2.

Urine Report. Amber; specific gravity, 1.025; reaction acid; no albumin; no sugar; urea, 1.6 per cent.

Microscopic Report. Negative.

Blood Report. Erythrocytes, 4,650,000; leucocytes, 10,200; hemoglobin, 70 per cent.

Because of the history of long-standing disease of the right ear, and because of a possible brain abscess, it was thought wise to subject the ear to thorough surgical exploration. Dr. Charles K. Mills, who saw the case in consultation at this time, fully agreed with me as to the advisability of this procedure.

Dr. de Schweinitz now made another eye examination, and reported as follows:

Mr. W. has a paresis of the right external rectus. He has double optic neuritis with many hemorrhages of the succulent and inflammatory type. Beyond this the examination revealed nothing new.

On May 12, 1902, the patient was operated upon by Dr. J. Chalmers da Costa.

A two-inch semilunar incision was made over the right mastoid, all structures being divided to the bone. The cartilaginous aural canal was then separated from the bony wall, and the mastoid chiseled. This was found decidedly hard and completely ossified; the opening was enlarged with rongeurs, and the middle and posterior fossæ of skull were exposed and explored. The dura

and brain tissue were found apparently normal, and hence the dura was not opened; the parts were thoroughly cleaned, hemorrhage controlled by torsion and ligature. A small strand of iodoform gauze was inserted, the aponeurosis was closed with catgut and the skin with silkworm gut. The drainage was removed at the expiration of forty-eight hours.

The patient made an uneventful recovery from the operation, and for a time was better, being free from headache, vertigo or vomiting. On May 20, however, in the morning, he complained of some vertigo and nausea. About noon had his wound dressed; suffered from nausea. Induced vomiting by means of his finger. Felt some relief after this vomiting, though the nausea persisted more or less for the remainder of the day. In the evening complained of a slight headache.

On May 25, 1902, he was re-examined by Dr. Mills and myself.

Station. A slight increase of sway.

Right knee-jerk slightly diminished; left knee-jerk slightly diminished.

Right ankle clonus slight disappearing; left ankle clonus, zero.

Right plantar reflex, zero; left plantar reflex, normal.

Faint hypesthesia of the inner aspect of lower portion of right thigh and right leg.

June 1, 1902. Symptoms as before. Tested for astereognosis upon the soles of the feet, it was found that he was somewhat uncertain as to the position of a pencil when the latter was placed in various positions upon the sole of the right foot. In one instance he clearly mistook the direction for transverse when it was longitudinal. Upon the sole of the left foot he made no errors whatever. Further, he did not recognize little balls of paper placed between the toes of the right foot as readily as when they were placed between the toes of the left foot.

June 3, 1902. Re-examination by Dr. Mills and myself.

Right knee-jerk, minus; left knee-jerk, minus.

Right ankle clonus, slight disappearing; left ankle clonus, zero.

Right plantar reflex, sometimes absence of response; left plantar reflex, normal. Other symptoms as before.

June 7, 1902. This evening (about 7.30) complained of nausea and vertigo. This attack appeared rather suddenly and was quickly followed by an attack of vomiting. Vomiting was not followed by any relief, though it recurred at intervals; it was accompanied by considerable retching. The vomited material consisted of a thick viscid mucus and some bile. Nausea and vertigo had almost disappeared by the following morning, after patient had slept fairly well.

June 10, 1902. Examined by Dr. W. G. Spiller. Present, Dr. Mills and myself.

Left knee-jerk, minus; right knee-jerk, more diminished.

Sensation for touch preserved in each lower limb and appears to be equal upon the two sides. The same is true of pain sensation. If hypesthesia is present upon the right side, it is exceedingly slight.

Movement of the toes to irritation of the sole of the right foot is that of flexion of all the toes, except the great toe; the great toe does not move. Upon the left side the movement is that of flexion, including that of the great toe. The only difference between the two sides is that the great toe of the right side does not move at all.

Left ankle clonus, zero; right ankle clonus, zero.

The Achilles jerk is prompt upon the left side, while upon the right side, tapping the tendo-Achillis causes repeated contractions, like ankle clonus.

Patellar clonus is not present upon either side.

Resistance to passive movements seems to be fully normal in each limb.

He stands erect without swaying, even when eyes are closed. The gait is not peculiar.

Hemiasynergy is not present in either lower limb when the patient is reclining.

The grasp of each hand is good and apparently normal.

The biceps jerk, triceps jerk and wrist reflexes are not very distinct upon either side.

Sensation to touch and pain in the upper limbs is equal upon the two sides, and is normal. States that his right hand is numb, that this is so distinct that he is certain it exists and yet upon testing it cannot be determined.

The patient states that since the operation, or possibly before the operation, he has been a little weak in the right lower limb, and also in the fingers of the right hand, arm and right leg; he complains of being awkward with the right hand. There is apparently some disturbance of sense of position. On attempting to put the first finger of the right hand on the first finger of the left hand, or vice versa, when eyes are closed, he has much difficulty in touching the desired objects. He is able to tell correctly any movement of his fingers or toes when his eyes are closed. Stereognostic perception is normal in both upper limbs.

Hemianopsia is not present. The pupils are equal. There is no nystagmus in looking to either side. Movement of the facial muscles is normal upon either side. Movements of the muscles of mastication are normal upon either side. There is no word-deaf-

ness, no word-blindness, and no symptom of sensory or motor aphasia. His memory is not quite as good as formerly. Says that he has become more irritable than formerly. Also that within the last twelve months he is not as good at figures as formerly; he cannot add or multiply as well as formerly, and this change he regards as very perceptible, and is even greater than the failing of memory. He has more difficulty than he formerly had in understanding the meaning of what he reads.

The patient is not as able to tell whether a pencil is placed lengthwise or crosswise upon the sole of his right foot; he makes no error upon the sole of the left foot.

June 20, 1902. Re-examined by Dr. Mills, Dr. Spiller and myself.

Lower limbs are well developed, although he says he is not as stout as a few years ago, weighing about twenty pounds less at the present time.

Resistance to passive movements of the thigh and leg, of each side, is normal and equal on the two sides.

Sensation to touch and pain in the lower limbs normal, though possibly tactile sensation is slightly diminished in the right lower limb as compared with the left. This diminution, if present, is slight. There is a slight diminution in the right lower limb to pain, tactile and temperature senses.

The patellar reflex on the right side is possibly slightly diminished; on the left side a little more pronounced than on the right side. Ankle clonus not obtained on either side. Plantar irritation upon the right side produces a slight movement of all toes in flexion, except the great toe, this not being moved at all; movement of the other toes is slight. On the left side, the movement of all the toes is that of flexion.

The Achilles jerk is present on each side and is about normal.

The gastrocnemius reflex is present on each side. These reflexes, Achilles and gastrocnemius, being equal on both sides.

Station erect is good with eyes closed. Gait normal. Stands well on each foot alone. Resistance to passive movement in the right upper limb is normal; the same in the left upper limb.

Sensation to touch, pain and temperature is normal in the two upper limbs. There is possibly a slight diminution in the right arm, but this is questionable.

Biceps jerk, triceps jerk and wrist reflexes are about normal upon the two sides. The sense of the position of the fingers and toes of each side seems to be normal, but he is not always certain of the right great toe. He seems to be able to tell correctly movements of the left great toe. Apparently there is no loss of the sense of position in the upper limbs, or at least the loss is not

pronounced. The movement of the right upper limb, when the eyes are closed, would suggest a slight loss of the sense of position.

The stereognostic sense is well preserved in both hands, although he occasionally makes a mistake in trying to differentiate between a dime and a cent with the right hand. These mistakes are infrequent. He never, however, makes any mistake with the left hand. Upon the sole of the right foot, there is again noted, as at previous examination, a tendency to make errors as to the position of the pencil or penknife when placed upon the sole of the right foot. Upon the sole of the left foot no errors are made at any time.

The patient was re-examined July 5 and July 29 and presented no noticeable change of symptoms. On September 5, 1902, he was re-examined by me. His symptoms were the same as at previous examinations, save that there was present an area of undoubted tenderness over the skull in the left post-parietal region. This area in front begins at eight and a quarter inches from the glabella and about half an inch from the sagittal suture. It extends as far back as to within one and three-quarter inches of the occipital protuberance. It is about two and three-quarter inches in its longitudinal diameter. It extends downward over the parietal region for about two inches. It appears to be oval in shape. The patch is sensitive upon superficial pressure and made worse upon deep pressure, and pain is also readily elicited by friction. The patient states that this area has been more or less noticeable since August 31.

Of late the patient has suffered but little from headache, having had but one severe attack since he was last examined. His diplopia is about the same. Dr. de Schweinitz reports his vision as perhaps slightly better. There is little or no change in the paresis of the left external rectus. There is less swelling of the optic discs than there was.

Because of the new symptom of tenderness in the post-parietal region, I submitted the patient to another examination on September 11, at which Drs. Mills and Spiller were also present. Strange to say this symptom of tenderness had at this examination almost or completely disappeared, so that it could not be confirmed by either of my colleagues. Otherwise the symptoms were as noted at previous examinations. It was thought by Drs. Mills and Spiller that possibly there was a slight awkwardness in the movement of the fingers of the right hand and of the toes of the right foot. Difference in the movement between the digits of the two sides of the body, however, was so slight as to be open to discussion. This remarkable symp-

tom of tenderness, both superficial and deep, upon the post-parietal region never subsequently recurred, although new symptoms made their appearance, while some of the old ones gradually became more pronounced.

On October 17, 1902, he again presented himself for an examination, and it was now found that in addition to the symptoms which he had presented before, there was a slight hypesthesia of the left side of the face. It was most marked over the middle distribution of the trigeminal and to a less extent externally over the superior. Mr. W. was able to detect all tactile impressions directly, but hypesthesia was undeniably present. He also described a subjective sensation of numbness and a crawling sensation in the left side of the face; also that the left side of the face felt somewhat stiff. The slight difference in the nasolabial folds, noticed in the early history of his case, was still present, but had not become accentuated. There was no involvement of the muscles of mastication. The patient had had very little headache, and only a slight attack of nausea some two weeks ago. About this time his case was studied by Dr. William Osler, who, with myself, thought that this numbness of the face suggested a deep basic lesion upon the left side. Dr. William M. Sweet now made several careful skiagraphs of the head, but no shadows were revealed that seemed in any way to suggest the location of a tumor. After a consultation with Drs. Mills and Spiller, it was determined to advise an exploratory operation of the base upon the left side. The patient's symptoms were at this time gradually becoming more pronounced, although the general symptoms were as before. The optic neuritis was now of high grade, with beginning degeneration of the optic nerve fibers, with resulting depreciation of vision (de Schweinitz). The fields were contracted, but there was no hemianopsia, the pupillary reactions were unchanged. There was a palsy of the right external rectus, thirty degrees, and a palsy of the right superior rectus (possibly inferior oblique) of eleven degrees. There was some widening of the palpebral fissures, giving the appearance seen in hydrocephalus.

On November 3, 1902, Dr. Keen made an osteoplastic flap in the left temporal region, but beyond determining a distinct resistance posteriorly, the operation resulted negatively as regards localization of the growth. Mr. W. made a perfect recovery from the operation and subsequently returned to his home. He was not seen again until December 17, 1902, when it was found that the hypesthesia of the left side of the face had disappeared. There was, however, a slight, but unmistakable hypesthesia present in the lower limb and to a less extent of the right upper

limb. The plantar reflex was not obtainable upon either side. The knee-jerks presented the same difference as at previous examinations, as did also the ankle clonus. The grip of the right hand was 65; that of the left hand, 60. The patient stood well upon either leg alone. The tongue was protruded in the median line. There was no facial inequality. The atrophic changes in the optic nerve were somewhat more marked than at the previous examination. When tested for astereognosis, occasionally made errors with the right hand. No word-blindness. No word-deafness.

The patient now passed from under my observation. I did not again see him until March 18, 1903. Dr. T. Percival Gerson, of Lansdowne, under whose care he was, reported that he had gradually become hemiplegic, there being a very gradual loss of power making its appearance in the right side of the body, right leg and right arm, also that there had been very decided loss of sensation upon the right side and awkwardness of movement.

March 18, 1903. Examination by Dr. Dercum, together with Dr. Gerson.

Mr. W. presented the following symptoms: He entered the room with a decided hemiplegic gait, dragging the right leg and allowing the right arm to hang at his side. He was not able to stand upon the right leg alone, while the loss of power in the right arm was also very marked. There was a decided hypesthesia of the right arm, right leg, right side of the face and head and right half of the trunk. This hypesthesia was not sharply demarcated by the middle line. It was most marked in the distal portions of the extremities and became less marked as the middle line of the trunk was reached. The area of but slightly impaired sensation extended from two to four inches from the middle line to the right. The right knee-jerk was much exaggerated. The left knee-jerk was about normal. There was now persistent ankle clonus upon the right side. The elbow jerk was present upon the right side, but not pronounced. There was a slight von Bechterew reflex upon the right side.

The Babinski reflex was not present upon either side. Upon the left side the toes moved normally, but on the right side there was a slight movement of the four outer toes in flexion, while the great toe did not move at all. Right-sided homonymous hemianopsia was now present, although both visual fields were distinctly contracted. This hemianopsia could readily be demonstrated and was quite well defined by the middle line. Wernicke's symptom was not present. Astereognosis was now complete. There was also typical word-blindness. There was no word-deafness. The patient had, however, a distinct difficulty in the use of substantives. He was unable frequently to give the name of

common objects, such as a book, even though he examined the book both by vision and touch. The symptom of anomia was not complete, but it was undoubtedly present. At this examination, tenderness in the post-parietal region was again sought for, but not elicited.

Because of the new features presented by the case, I decided to again advise an operation, and submitted the question to my colleagues, Drs. Mills and Spiller, who concurred in its advisability. Accordingly, on March 26, he was operated upon by Dr. Keen, who made a large exposure of the parietal region. The details of the operation are given by himself. The bone proved to be excessively vascular, and the operation had to be completed in three stages, the final result being the removal of an enormous sarcoma, weighing 264 grams. The mass was encapsulated and was removed without much difficulty, but the patient succumbed to shock and operation shortly afterward.

Although no autopsy was obtained, the fact that the tumor was encapsulated makes it exceedingly probable that only one sarcomatous mass was present in the brain, and that all of the symptoms were referable to this growth. The interest of this case lies in the extreme difficulty of the localization and in the indefinite and often misleading character of the symptoms. It is remarkable that cortical disturbances, such as unmistakable astereognosis and alexia on the one hand and hemianopsia upon the other, were not present earlier in a case of tumor in this situation. The inference is justified that the growth originated subcortically and only late in the course of the affection produced cortical phenomena by upward growth and hemianopsia and basal phenomena by downward growth. In reviewing the symptoms we are impressed by the fact of the insignificant value of such a sign as a paralysis of one abducens or of a trifacial hypesthesia. In the present instance they were undoubtedly due to pressure from a distance, and as is well known, they frequently so occur from such cause. They are of themselves of little or no localizing value. However, the case does demonstrate the localizing value of astereognosis and slight muscular incoordination. In reviewing the history of the case, it will be noted that faint, but unmistakable astereognostic signs were present upon the sole of the right foot, and somewhat later upon the right hand, as were also faint, though somewhat inconstant

symptoms of incoordination of the right fingers and right toes. Lastly, the symptom of local tenderness and pain in the post-parietal region, which was present for a few days only, assumes a vast importance. It was so fugitive that it was not present at the examination made a week later by Drs. Mills and Spiller, and was apparently absent at the examination made by Dr. Osler, and yet that there was abundant cause for such a pain, the finding at the operation of a large exostosis of the internal table in this very situation most conclusively proves.

To repeat, the history of the case and the very gradual evolution of localizing phenomena justify the inference that the tumor began subcortically in the post-parietal region, and that it grew downward and upward very gradually. It would seem to me that it would be a perfectly justifiable expedient and far preferable to what was done by ourselves in this case, when symptoms of one-sided trouble, vague in character, make their appearance, not to attempt an exploration of the base, which is always a matter of extreme difficulty, but to make a free osteoplastic exposure of the most probable area upon the lateral aspect. The free osteoplastic exposure is attended with little risk, and the exploration possible under the circumstances is, as a rule, very thorough. Had such a plan been attempted by us I am quite sure that the tumor would have been found, though early in the case it would doubtless have been found to be situated beneath the cortex. The lesson to be drawn from this case is to make free osteoplastic exposures in all cases yielding symptoms referable to one side of the cortex, no matter how slight these symptoms may appear to be.

The tumor apparently grew very rapidly in the last few months of the patient's life. The size and weight that it attained were truly enormous, and showed how very much pressure the brain can withstand without destroying the life of the patient.

Surgical Report by Dr. W. W. Keen.

I first saw Mr. White in consultation with Dr. Dercum on October 29, 1902. The reasons for operating at the point selected, and also for the site of the prior operation which Dr. DaCosta made on the right side in the spring of 1902, have already been given by Dr. Dercum.

Operation November 3, 1902. I made a flap in the left temporal region (Fig. 1) practically the same as that for the Gasserian ganglion, including chiseling the zygoma so as to get down as low as possible. First, with a gouge I made a small opening in the bone, and then with the rongeur forceps enlarged it till it was 4 to 5 cm. in diameter. The bone seemed to be unusually thick, two or three times the ordinary thickness in this position. Exposure of the dura showed that it was very resistant and bulged considerably. As soon as it was opened the brain protruded. I attempted to lift the temporo-sphenoidal lobe with a broad spatula, so as to gain access to the pons, where the tumor, it was thought, might possibly be. It was, however, evident that the protrusion of the brain was such that I should do very extensive damage to the brain tissue if I lifted it to the desired extent. The cortex did not seem harder than usual, though the sense of resistance in the brain as a whole was quite marked.

In order to determine whether there was any possible distension by fluid, I punctured the lateral ventricle, but did not find any. The diminution of resistance when the ventricle was entered was very perceptible. I then incised the temporo-sphenoidal lobe to a depth of 3.5 cm. and introduced my finger into the incision. The white substance was normal to the eye. To the finger there was a distinct increase of resistance to pressure *posteriorly* as compared with the other three directions. After making several attempts to lift the temporo-sphenoidal lobe, and failing to see even as far as the site of the Gasserian ganglion, I abandoned the operation. He made a perfectly smooth recovery, the highest temperature being 99.6 degrees.

Second operation, March 26, 1903. First Stage. Owing to the development of new symptoms, especially hemianopsia, after a consultation with Drs. Dercum, Mills and Spiller, we decided that it would be wise to do a second operation. The site of the tumor was now more accurately located in the parietal lobe extending forward perhaps as far as the fissure of Rolando and well backward into the occipital lobe. It will be observed that when at the first operation I introduced my finger into the substance of the temporo-sphenoidal lobe, I recorded at that time that the resistance posteriorly was distinctly greater than in the other three directions. This also would confirm this diagnosis as to the situation of the tumor.

I outlined a flap, the anterior border of which crossed the fissure of Rolando about at its middle, and passed nearly vertically upward, 9 cm. long (Fig. 1). Parallel with the median line, but 2.5 cm. away from it, I made an incision 13 cm. long; this reached into the occipital lobe. The posterior incision was

12 cm. long, as indicated in the diagram. The hemorrhage from the scalp was exceedingly profuse. At every point where the bone was exposed sufficiently to chisel it, wherever a little vessel penetrated the skull, the blood poured out from the bone in a fountain nearly 1 cm. in height. These were stopped by Horsley's wax, the vessels in the scalp being clamped. I then proceeded to chisel the bone. Of course the incision was not all made at first, but in three sections, and the chiseling of one was completed before the next was begun. The bone also I found very vascular. Some of the veins of the diploë poured out distinct streams of blood. These also were stopped by Horsley's



Fig. 1.

wax, but in spite of this a large amount of blood was lost. I could not understand why, especially at the posterior portion of the median incision, I did not get through the bone. While I was operating, one of my assistants, Dr. Craig, opened a vein in the right arm and infused 20 ounces of salt solution, to which were added 20 drops of 1-1000 adrenalin solution. Finally, as the patient had lost so much blood, we decided that the vascularity of the parts showed that the tumor lay underneath the proposed flap, that it was a very vascular sarcoma, and that if I continued the operation there would be so large a loss of blood

when I turned back the osteoplastic flap that it would probably cost him his life. I decided, therefore, to do the operation in two stages.

After the operation his temperature, up to the 29th, rose to a little above 100° , but from the 29th to the 31st fluctuated between 101° and 102.6° . In spite of the high temperature, he did not seem to be very sick, though his pulse was from 100 to 120.

Second stage. On March 31 we decided to reopen the skull and operate on the tumor. As I anticipated, a very severe hemorrhage from the tumor, I first clamped the common carotid with Crile's clamp and obliterated the caliber of the artery. After reopening the wound in the scalp, which was attended with very free bleeding again, I chiseled some places in the bone where I had not chiseled entirely through, especially in the portion parallel with the median line just posterior to its middle, and at last was able to turn back the flap. As soon as it was reflected, the difficulty of chiseling through the bone was explained. On the inner surface, just at the median edge of the flap and 3 cm. from its posterior end was a bony tumor, conical in shape, 2.5 cm. in height and 2.5 cm. in diameter. The chisel had traversed this bony tumor, where the bone was about two to three times its ordinary thickness. With the rongeur forceps I removed the entire bony growth. The dura was very soft, and at one point there was a deep depression corresponding to the bony growth. The tumor was evidently partly cystic. I then opened the dura corresponding to the osteoplastic flap. A considerable amount, estimated to be seven and one-half ounces, of bloody serum, so bloody that at first I thought it to be pure blood, escaped. This led me to ligate the common carotid and remove the clamp.

As soon as the brain was exposed, the entire opening in the bone, large as it was, was filled with a very large bulging sarcoma. It was so large and his condition was so critical, in spite of another infusion of a pint and a half of salt solution with 30 drops of adrenalin 1:1000 solution, that my judgment was against any attempt to remove the entire tumor, but I scooped out with my fingers a large handful. The hemorrhage was free, but not alarming. Packing with iodoform gauze, douching with hot salt solution checked the bleeding, and I closed the wound after gnawing away a part of the edge of the bony flap in order to allow the end of the gauze packing to protrude. I also ordered five minim doses of the adrenalin every four hours hypodermatically.

On the day after this operation his temperature rose to 103.2° and continued between 100° and 102° for nine days. It then fluctuated between the normal and 100° ; meantime the wound

had healed without incident. The packing was removed on the second day without any noticeable hemorrhage; the wound in the neck healed by first intention.

On April 1, the day after the operation, he moved his right arm once, but then was completely paralyzed on the right side for a week. Movement then began both in the right arm and leg, and he gained so rapidly that, after sitting up in bed for two or three days, I got him out on a chair, and on the seventeenth day he was able to walk down stairs into the garden of the hospital.

As his parents and he himself, appreciated the fact that if the tumor were not removed the end would be fatal, they consented to another and final attempt to remove the remainder of the growth. In view of his very good condition, Drs. Dercum, Mills, Spiller and myself, after a conference, determined upon the final attempt.

Third stage, April 21st. The flap was reopened very readily, the adhesions giving way under moderate leverage. The same peculiarity noticed before, was again evident, viz,—abundant hemorrhage not only from the scalp, but that every vessel which penetrated the bone became a little fountain. Again Horsley's putty stopped this quite effectually. After gnawing away the bone sufficiently and removing the diseased portion of the dura, I was able to reach the limits of the tumor. The tumor extended 2 cm. in front of the anterior border of my original opening in the bone, 1 cm. inferiorly and 1 cm. posteriorly. It extended in the middle line entirely to the falx. This large mass, which more than filled my entire hand, I was able to enucleate quite easily and cleanly. Fully one-half of the entire area of the falx was exposed as soon as the tumor was removed. The hemorrhage, especially from the middle and posterior portions of the wound was very profuse. Packing with iodoform gauze arrested it in part. Adrenalin was given together with a saline infusion continuously until 30 drops of 1:1000 solution of adrenalin and a pint and a half of salt solution had been administered. No distinctly large vessels, which could be ligated were discoverable. Accordingly I had to trust entirely to packing and hot water. The flow was so profuse that adrenalin locally would have done no good. The wound was packed as firmly as was deemed advisable, the wound closed and the patient put to bed. He was in very profound shock and died about half an hour after being placed in bed. A culture from the surface of the tumor was given to Professor Coplin, who reported that the micrococcus pyogenes albus in pure culture was found. No abscess

existed but the surface of the tumor was softened over a small area.

REMARKS.

Weight. This tumor is the largest that I have ever removed from the brain. I have not compared it with any accurate list to see what others have exceeded it in weight, but so far as I know the only one which exceeds it, is that removed, with better success, I am glad to say, by Bramann. His tumor weighed 280 grms., 16 grms. more than this. The tumor here reported weighed 264 grms., more than half a pound.

Hemorrhage. In few, if any, cases have I seen more severe hemorrhage from both scalp and bone. This seemed to indicate that the tumor lay directly under the site of the final operations. In the bone, Horsley's wax answered admirably in arresting the bleeding. At the final operation the hemorrhage from the brain was so great that even quite firm gauze packing did but little good. No known large sinus, vein, or artery was opened, but the hemorrhage was universal and severe. The application of Crile's clamp and finally ligation of the common carotid on that side modified it but little.

Exposure of the Falx Cerebri. Only very rarely have I removed a tumor which exposed the falx. In this case the exposure covered at least one-half of the superficial area of the falx, though less than one-half of its length.

Dr. Spiller examined the tumor and made the following report:

"At the earlier operation by Dr. Keen, a large portion of the tumor was removed. The mass removed was easily broken, and had in the fresh state the appearance of a sarcoma. The portion removed was hardened partly in formaline and partly in Müller's fluid. After hardening had occurred the portion in formaline weighed 27 grms., that in Müller's fluid 49 grms. The part first removed has an irregular surface as though it had been torn away from a tumor mass.

"The part of the tumor removed at the later operation is much larger than that removed at the earlier operation, and two-thirds of its surface are sharply defined and nodular, as though it had been sharply separated in this portion from the surrounding brain tissue. Approximately one-third of the entire tumor, if one may judge from the form of this large tumor, must have been removed at the earlier operation. One side of the large tumor has a torn appearance, and from here probably the first piece of tumor was removed. The upper surface of the tumor is flat. The

weight of the second portion removed, before any hardening fluid had been used, was 188 grms., so that the entire weight of the tumor, including hardened and unhardened tissue, was 264 grms.

"The tumor consists of small spindle-shaped cells without definite arrangement, and is not exceedingly vascular. It is a small spindle-cell sarcoma."

STUDIES UPON THE CEREBRAL CORTEX IN THE NORMAL HUMAN BRAIN AND IN DEMENTIA PARALYTICA.

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(Continued from page 716.)

Occipital Region.—Coming to the posterior pole of the external cortex, the typical structure of the occipital lobe is represented by Plate VIII, Fig. 24, a section magnified 14 diameters from a block taken from the point indicated by the figure 8, Plate I, Fig. 1. The section is 6-3 microns in thickness, is 6 mm. in width at the widest portion, and 7 mm. in length, stained with methylene violet, fixed in Van Gehuchten's fluid, other technique as previously described. This convolution is seen to be quite different in shape from the two previously described of the temporal and parietal regions, having a broader base and narrower rounded vertex. The cortex is seen to be thickest on the lateral aspect *b*, thinner on the opposite lateral aspect *c*, and thinnest at the vertex in the vicinity of *a*; the central white medullary substance being reduced to a minimum. The outer layer varies in thickness, being thickest at *c*. Above this it is broken away for a short distance, is quite thin at the vertex, and becomes somewhat thicker on the lateral aspect *b*. Above *b* the section is seen to be cracked, an artefact resulting from its fixation upon the slide, also at *c* numerous small breaks are noticed, due to the microtome knife. Opposite *a*, and enclosed in ink lines, is the segment of this section, represented in Plate IX, Fig. 25, under a magnification of 100 diameters. The radial arrangement of the cells is well made out at the vertex, but not quite so readily seen on the lateral aspects of the gyrus here, but under a higher magnification can be distinctly made out. About the middle of the second layer, especially at the vertex, some very large pyramidal cells are to be seen singly or in groups, and approaching the Betz cells in type in some cases. The third or lower layer presents nothing unusual, and disappears below into the white medullary substance of the interior of the gyrus. Plate IX, Fig. 25, is a photomicrograph of the segment *a*, enclosed in ink lines in Plate VIII, Fig. 24, at a magnification of 100 diameters. Here the cortex is found thinner than in any of the preceding plates, measuring scarce 2 mm. in thickness. The first layer averages only .15 mm. in thickness, and

in structure and arrangement of the cells is similar to this layer in the preceding plates. The second layer measures 1.15 mm. in thickness, containing small pyramidal cells above and quite closely packed together, but increasing in size below and being somewhat more scattered. About the middle of this layer a number of large cells are seen, many singly, but some arranged in groups in places.

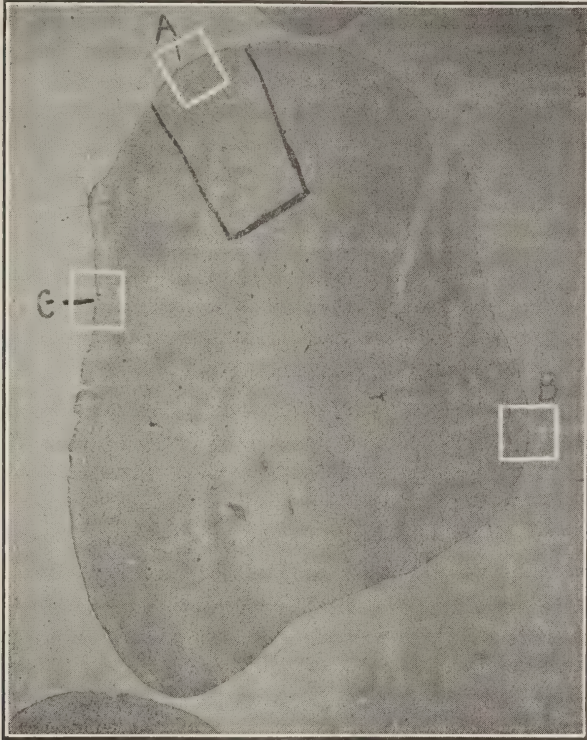


Plate VIII, Fig. 24.

Some of these are even larger than those found in the parietal convolution, and are intermediate in size between the Betz or giant pyramidal cells and the large pyramidal cells. These latter are not very numerous in this region, the small pyramidal cells making up by far the greatest part of the cellular elements of this layer. Below the middle of this layer is to be found a region, at *a*, almost exclusively made up of small pyramidal cells about .25 mm. in width. Between this and the lower border large pyram-

idal cells are again found intermingled with the smaller. The third or spindle cell layer is here .70 mm. in thickness, containing larger and smaller spindle and irregularly polygonal cells, the lower border being gradually lost in the white medullary substance below. The nerve cells are found quite numerous and closely aggregated in this region, as counts by the same method and manner as in all previous sections, give here an average of 164 nerve cells to the square millimeter of surface of the section, and 199 neuroglia cells, the section being 6-3 microns in thickness and stained with methylene violet. The cells are thus seen

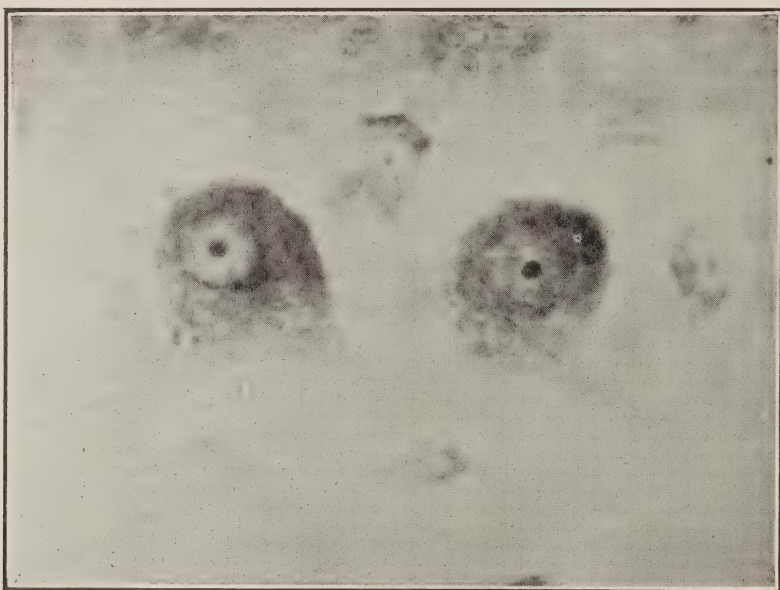


Plate IX, Fig. 27.

to be more numerous than in any other region of the external surface of the hemisphere, with the exception of the posterior central convolution. The fact of the greater number of the pyramidal cells being small, permits of their closer aggregation, and no doubt is an important factor in the result of the nerve cell counts. The number of neuroglia cells averages higher than in any other region posterior to the fissure of Rolando, but they are less in number than in the anterior central convolution.

This includes the plates of the external surface of the cortex, but for the purpose of comparison, several plates will be intro-

duced from other parts of the central nervous system. The first of these is a photomicrograph, Plate IX, Fig. 26, of several large pyramidal cells from the layer of large pyramidal cells of the hippocampus major or cornu ammonis; between the alveus, representing the white medullary substance of the ordinary gyrus, and the stratum radiatum containing the apical dendrite processes of

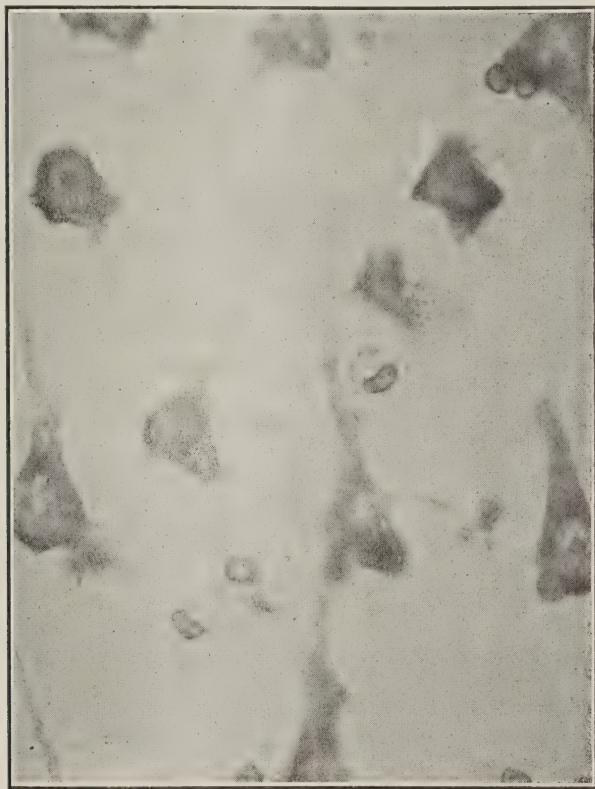


Plate IX, Fig. 26.

these hippocampal pyramidal cells. This plate and the two following are not presented as a basis of comparison with similar regions of the pathological brain subsequently to be described, but to give an idea of the appearance of these cells in a normal brain as seen in a photomicrograph of a Nissl preparation magnified 1,400 diameters, as distinct types in Nissl's classification.

These large cells of the cornu ammonis are given as one of the four types of Group II of the stichochrome nerve cells in his classification; the other three types being (1) the nerve cells of the motor nuclei, (2) certain cells of the cerebral cortex, and (3) certain cells of the spinal ganglia. In shape these cells approach the larger pyramidal cells of the external surface of the cortex, second layer, but are somewhat more slender, of a narrow pyramidal shape, have fewer basal dendritic processes, and a long, somewhat slender, apical dendritic process. The chromatic substance here is made up of very small granules fusing into larger and smaller masses in places, so that in the photomicrograph it is difficult to differentiate and determine their arrangement accurately. In some of the cells, as in the cell to the extreme right of the plate, and partially out of the field, the rounded and linear granules above the nucleus, and extending into the apical process, are seen to have a general parallel arrangement. The nuclei of these cells are large, and contain a large, well marked nucleolus and a more or less irregular chromatic network. As this section was stained with methylene violet, numerous neuroglia cells are distinctly seen scattered among the pyramidal nerve cells.

From the cerebellar cortex a photomicrograph was taken of two Purkinje cells, magnified 1,400 diameters, and shown in Plate IX, Fig. 27. The section from which this photomicrograph was taken was situated on the external surface of the left hemisphere, cut transversely to the horizontal axis of the same, fixed in alcohol 95 per cent, stained with methylene blue: other technique similar to that of previously described plates. The section is 10 microns in thickness. These Purkinje cells were given by Nissl as typical examples of what he formerly described as Group II, arkyostichochrome nerve cells of the somatochrome class, in which was presented a striated appearance, with a network-like structure, united in a most intricate manner, thus having characteristics of both the arkychrome and the stichochrome cells. He now classifies them as one of the types of Group I, the arkyochrome nerve cells. In the Purkinje cell, to the right, the base of each of the two large dendritic processes is seen, one going off to the right, the other to the left, subsequently to divide and subdivide in the internal layer, not shown in the plate. The Purkinje cell to the left shows only one of these processes in this plane, that going off to the left which divides in the same manner as above described for the other cell. The large rounded nucleus, with its centrally placed nucleolus, is fairly well shown in the cell to the right, and contains a fine network of chromatic substance. The chromatic substance of the body of the cell is made up of larger and smaller irregularly-rounded chromophilic bodies, arranged in an indefinite network extending up into the base of the dendritic

processes. A nuclear cap is seen above the nucleus of the Purkinje cell on the right. The closely-packed fine granular cells of the granular layer of the cerebellum are seen just below these two Purkinje cells, and are included in the class of cytochrome nerve cells in Nissl's classification. They contain a nucleus almost

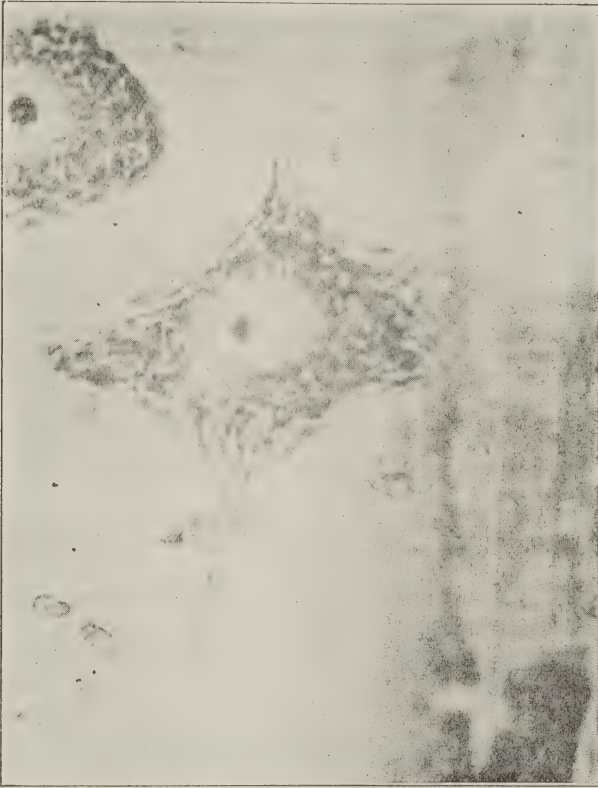


Plate X, Fig. 28.

filling the cell body, and which is surrounded by only a very narrow rim of chromatic substance. Within the nucleus is a nucleolus, and in some of these nuclei several irregularly-arranged chromatic granules are also seen. Between the Purkinje cells is a small nerve cell with rounded body and an apical dendritic process. It contains a nucleus, nucleolus, and some chromophilic granules irregularly arranged in the cell-body.

The last photomicrograph in the series of the normal histological material is that shown in Plate X, Fig. 28, of a typical multipolar ganglion cell from the anterior horn of the lumbar enlargement of the spinal cord, under a magnification of 1,400 diameters. The segment of cord from which the section was taken was fixed in Van Gehuchten's fluid; this section is 6 2-3 microns in thickness and stained with methylene violet. This cell is, according to Nissl's classification, an arkyochrome nerve cell, Group I, of the somatochrome class, the chromophilic bodies being arranged more in the form of a network than in a parallel arrangement in the cell-body, though within the dendritic processes these bodies are more or less distinctly parallel in arrangement. The cell-body is irregularly polygonal in outline, with five large dendritic processes going off in different directions, and containing at the base, and for a considerable distance, linear-shaped chromophilic bodies, arranged in a more or less parallel manner and spreading out and connected with the cell-body network. This latter is made up of irregularly-rounded and elongated chromophilic bodies, arranged in an irregular network. The nucleus is large, rounded, slightly eccentric in situation, and contains a large rounded nucleolus and a pale indefinite network of chromatic substance. Numerous neuroglia cells are seen scattered about, singly or in groups, in the immediate vicinity of this cell. Part of another large ganglion cell is seen at the upper portion of the plate, on the left.

This concludes the very brief discussion of the cortex of the normal brain, in which an endeavor has been made to present typical sections of the main divisions of the external surface of the cerebral hemisphere by means of photomicrography, upon which the greatest care was employed, in order to give an accurate idea of the usual appearance to be seen under the microscope in the study of such sections in contradistinction to the common diagrammatic figures that one usually associates with such work, and which do not show the actual arrangement and structure, but rather the ideal or embodiment in one figure of what is to be found in parts of many. The writer, on the other hand, does not wish to be understood as not favoring diagrammatic or schematic figures in work of this kind, as he regards them as highly important and a valuable accessory to photomicrography and accurate drawings in the proper conception of the intricate structure and arrangement of the various portions of the nervous system as well as other systems. That there are minor variations from these plates in the different regions has been repeatedly stated, but the variation is only a minor one, and does not depart sufficiently from the type to be considered as more than a modification. Furthermore, in going over the various plates from the

different regions it will be seen that there is a general uniformity of arrangement and structure of the cells, and that one region does not vary to any great extent from that of any other region. This is well illustrated by comparing the plates from the frontal region with those of the temporal region, in which it would be difficult to determine the one from the other. The Betz cells in parts of the motor cortex are a conspicuous modification, but when we consider that only a small part of the motor area contains these cells, and again that these cells, though large, represent but a minute fraction of the total number of cells in the cortex at this point it will be seen their presence makes but a slight modification in the general cortical arrangement as a whole. The pyramidal cells intermediate in size between the large pyramidal cells and the Betz cells are of especial significance to the writer as perhaps indicating that, with increased specialization of function, carried on through long periods of time, increased specialization of the structure and size of the cortical cells is going on hand in hand, and that perhaps the so-called Betz cells, now almost entirely limited to certain portions of the central convolutions, some time in the future may be found in all parts of the cortex. Their large size, with resulting large amount of protoplasm in the cell-body and large nucleus, the comparatively enormous amount of chromatic substance, as possibly stored up food products, all point to great capacity for storing up nervous energy to be given out when indicated as powerful efferent impulses, or, on the other hand, the capacity of receiving as equally powerful different impulses. Increased specialization in mental activity may result in greater numbers of nerve cells being set apart and increased in size and capacity for carrying out special functions.

Paretic Brain.—Turning now to Brain B, the case of dementia paralytica, from which all the succeeding plates were taken, we see in Plate X, Fig. 29, a photograph of the left hemisphere of the brain, natural size. The macroscopic appearance at first sight would seem to indicate that there was marked atrophy and shrinkage in parts of several convolutions, especially the anterior central and frontal convolutions; but a careful study of the sections taken from these regions shows this not to be altogether the case, or at least only to a slight degree. As any one who has studied brain topography to any extent well knows, there is much variation, not only in the shape and arrangement of any given convolution, but also in its size, so that great care should be observed in not mistaking such normal variation for atrophy. A study of the various plates shown here under a magnification of 14 diameters, containing the entire cross section from the several convolutions in the brain and comparing them to the preceding brain will make this point clear. Here and there a considerable quantity of the

cortex has been torn away with the pia mater, whereas in many places small depressions are seen in which only a very small portion of the superficial layer of the cortex has been torn away. The convolutions are here well marked and the sulci very deep, so that there is a large area of cortical surface. The sulci in the middle portion of the hemisphere are widely open, owing to the support being placed only in the middle of the median surface of the hemisphere, so that the weight of the poles, anterior and posterior, caused these latter to become depressed, resulting in this conspicuous widening of the central sulci. The posterior pole of the brain—the occipital lobe—is practically normal macroscopically,

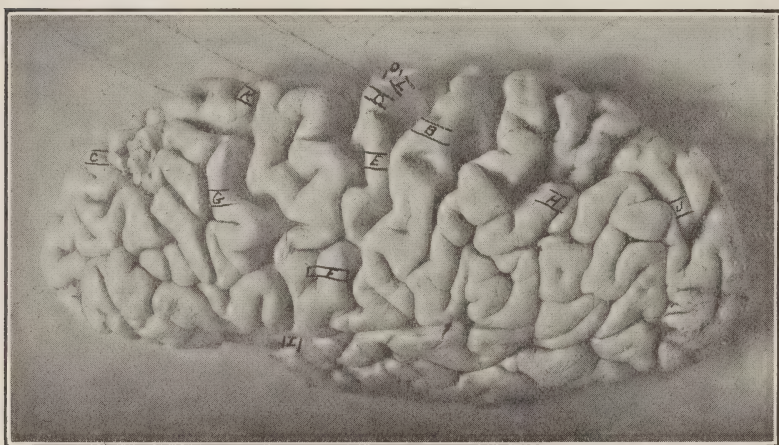


Plate X, Fig. 29.

and it will be seen later also microscopically, there being none or but very slight pathological changes in the cortex in that region. This photograph of the brain was taken before any blocks were removed, it having been previously fixed in 95 per cent alcohol. As each block was removed the exact location was noted and indicated in the photograph by the letters of the alphabet, and the place from which the block was taken enclosed in ink lines.

Frontal Convolution.—This portion of the cortex is represented by the block K, taken from the external surface of the first frontal convolution in its middle third, the exact location being seen in Plate X, Fig. 29, K. The photomicrograph seen in Plate X, Fig. 30, was taken from a section 10 microns in thickness, and is magnified 14 diameters. The gyrus here is somewhat narrow, and the sulci on either side were quite open and deep, and from

the macroscopic appearance one might at first be inclined to think that there was some atrophy of the gyrus; but a detailed study of sections from this region shows that this is not the case. Block K was fixed in 95 per cent alcohol; this section is 1 cm. in length, .63 cm. in width at its widest part, and, as above stated, 10 microns in thickness, being stained with methylene blue; the other technique being the same as for all the other plates. The shape of the section is somewhat that of a truncated pyramid, broader at the base and narrow at the rounded vertex. This me-

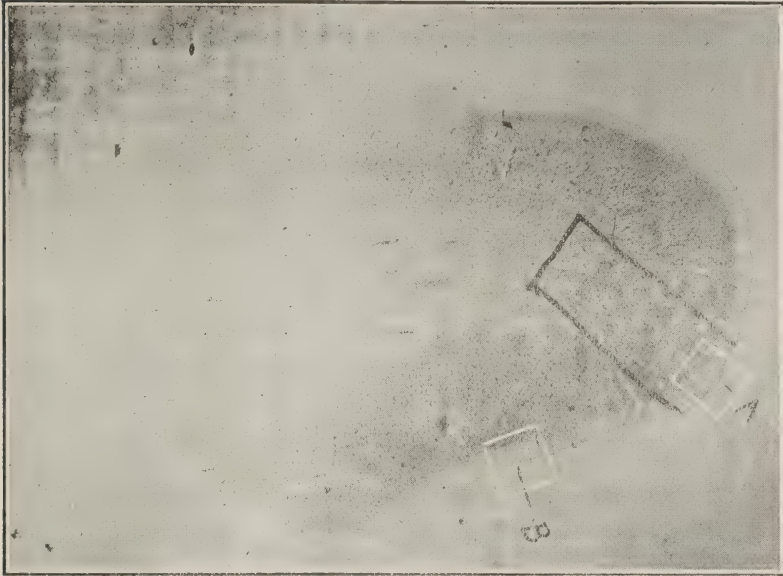


Plate X, Fig. 30.

chanical arrangement makes the cortex thicker at the vertex than on the lateral aspects. At *a*, and enclosed in ink lines, is the segment represented in Plate XI, Fig. 31. The first or superficial layer is seen to vary somewhat in thickness, being thicker on the lateral surface below *b*, with another thickened portion below *a*, a third just to the left of the vertex, and finally at the lower part of the left lateral aspect. The second or pyramidal cell layer does not show quite as distinct arrangement of the cells in a radial direction as in the corresponding region of the normal cortex, and there is especially to be noted the numerous portions of minute capillaries of varying size and tortuosity, singly or

branched. This second layer passes over into the indistinct third layer, which is lost below in the pyramidal-shaped white medullary center, which also contains numerous capillaries. Plate XI, Fig. 31, is a photomicrograph, magnified 100 diameters, of a segment corresponding in position to *a* of a section adjacent to and from the same block as that shown in Plate X, Fig. 30. The section is 6-2-3 microns in thickness, and of the same length and breadth as that shown in the preceding plate. Decolorization has been carried on to a considerable extent, about to the same degree as in Plate II, Fig. 3, and thus causing the plate to appear rather pale. Additional causes are the paucity of large pyramidal cells in the second layer, and the shrunken condition of many of the cells, as well as other pathological alterations. The upper layer averages .20 mm. in thickness, and contains for the most part scattered neuroglia cells. The second or pyramidal-cell layer is 1.50 mm. in thickness. Here is to be found a very similar arrangement of the cells to that in the corresponding region of the normal brain A. In the upper portion are to be seen the small pyramidal cells in considerable numbers, just below the superficial layer. Then they become more scattered, with a tendency to an arrangement into larger and smaller irregular groups, the cells becoming larger in the deeper portions. A little below the middle of this layer is found a narrow strip at *a*, where the small pyramids predominate almost to the entire exclusion of the larger ones, which latter, however, are seen above and below. Below this again are to be seen the larger pyramids, which finally give way to the irregular and spindle cells of the third layer. The nerve cells themselves show various changes. Many of them appear to be atrophied or shrunken, this process varying in degree in different cells. This in many cases has resulted in the formation of larger or smaller pericellular spaces as seen about many of these cells, as at *P* in this plate and in Fig. *P* of the text, for instance. Here a large pericellular space completely surrounds the cell. The basal dendrites are represented by only one small shrunken process given off at the left and extending but a short distance. The body of the cell is shrunken and irregular in contour. A large mass of yellowish pigment is seen at the base to the right. A small amount of finely granular chromophilic substance is found about and above the nucleus. The nucleus itself is displaced downward into the base of the cell-body, to the extreme left. It is small, irregular in outline, and contains a well defined nucleolus but no chromatic substance. The apical process is shrunken and irregular in direction, and contains traces of chromophilic substance in places. The chromatic substance in the large majority of these cells has become diffused and decreased in amount in variable degrees, so that a large number of the cells

present the appearance of chromatolysis up to almost complete disappearance of chromatin in some of them. These latter present a pale, washed-out appearance, as shown by the cell marked Q in this plate and seen enlarged in Fig. Q, where only a small amount of chromatin is found in the apical process and a slight amount of pigment in the lower part of the base. Some cells show loss of chromatin only about the nucleus, and are spoken of as instances of central chromatolysis. Others have a disappearance of chromatin at the periphery of the cell-body only, and this condition is known as peripheral chromatolysis. The nuclei of many of these cells are found frequently displaced to one side, crowded into the base of a dendritic process, near the base of the cell-body, or near the base, or up into the proximal portion of the apical process. Many of these are irregular in shape and diminished in size. Quite a large proportion of the larger pyramidal cells contain light yellowish pigment in variable quantity and usually at the base of the cell, often extending into the base of a dendritic process. The cell P, above described, in addition to the pericellular space, demonstrates these last two points, the nucleus being crowded into the base of the dendritic process given off on the left and a considerable deposit of pigment being found at the base and extending into the right dendritic process. The cells containing this pigment are marked with a cross, adjacent to them, in this plate. It will be seen that they are to be found mostly in the middle and lower portion of this layer, and also some few scattered irregular-shaped cells in the third layer. The dendritic processes in many of these cells end abruptly beyond their base, and in but few cases can they be traced to any great distance from the cell-body. The apical process shares in this general atrophy, and often is curved and irregular in direction, instead of presenting a regular straight course towards the periphery of the cortex. In some cells the nucleus is difficult to distinguish, the limiting membrane being indeterminable and the nucleolus appearing to be in the midst and surrounded by only the cell-body, and usually in an eccentric position. Numerous blood vessels are here to be seen, all with thickened walls and pursuing a tortuous course. Large perivascular spaces are also seen about many of these blood vessels, as at *d*, for instance. The third, or spindle or irregular cell layer is here .90 mm. in thickness, and is made up of irregular and spindle cells, some of the former, as some of the cells of the second layer, contain pigmentary deposits. Here also the chromatin is found in diminished quantity, and various stages of chromatolysis are seen. The nucleus is diminished in size in many cases and does not appear to be as prominent a factor as in the normal cells of this region. The cells as a whole appear smaller in size and more or less atrophied, containing less proto-

plasm in the cell-body as well as a diminished amount of chromatin. The blood vessels are here also found to have thickened walls, and are tortuous in direction. At *e* is seen one of these vessels, with thickened walls and surrounded by a large perivascular space. The entire thickness of the cortex at this point is found to be 2.90 mm.; almost the same as in the corresponding region in Brain A, as seen in Plate II, Fig. 3. Counts were made of the nerve and neuroglia cells in various parts of this section in the same manner as in the previous sections, and in eight different fields of 36 sq. mm. each of the ocular net-micrometer from various portions of the two lower layers for the nerve cell counts, and in all three layers for the neuroglia cell counts. There was found to be an average of 100 nerve cells and 56.90 neuroglia cells to each square millimeter of surface of this section. The small number of neuroglia cells being due to the methylene blue stain employed and differentiation carried on to a considerable degree in a section 6 2-3 microns in thickness. In the section shown in Plate X, Fig. 30, 10 microns in thickness, and more deeply stained, the number of nerve cells is not quite so great, whereas the number of neuroglia cells is almost six times as great, although the two sections are quite near together and from the same block. This is due to the neuroglia cells in Plate XI, Fig. 31, being almost completely decolorized in the greater differentiation, whereas the cells, although paler, yet were not decolorized, and can all be made out. The same method of using the ocular net-micrometer was employed here as in all the other plates, and counts were made from eight different fields of thirty-six squares each from different parts of the second and third layers of this section, and the average number of nerve cells was found to be 82.91 to the square millimeter of surface of the section, as compared to 100 for the same area in Plate XI, Fig. 31, so that there are on an average less nerve cells in this section, 10 microns in thickness, than in the section from which Plate XI, Fig. 31 was taken, which was but 6 2-3 microns in thickness, the same stain being used in both cases. The nerve cells, however, are more deeply stained in this latter section, and appear more distinct, and there are also more large cells. The neuroglia cells in eight different fields of thirty-six squares each from various parts of the three cortical layers average 303 to the square millimeter of surface of the section, as contrasted to 56.90 to the square millimeter in Plate XI, Fig. 31. Here increased thickness makes a difference in favor of this plate, and in addition to that the deeper stain with less decolorization cause all the neuroglia cells to appear more prominent. As stated before it was found that sections from 6 2-3 to 10 microns in thickness contain all the nerve cells to be seen in one plane. As the neuroglia cells are much smaller in diameter and

often arranged in dense clusters, there may be a greater number in a section 10 microns in thickness than in one 6-2-3 microns in thickness. (See Table II.)

Central Region.—Turning to the region posterior to the one just discussed, we come to the central region, or motor area, made up of the anterior and posterior central convolutions. Typical of the anterior central convolution in its upper portion is the section represented in Plate XI, Fig. 32, under a magnification of 14 diameters. The exact location of the block from which this sec-

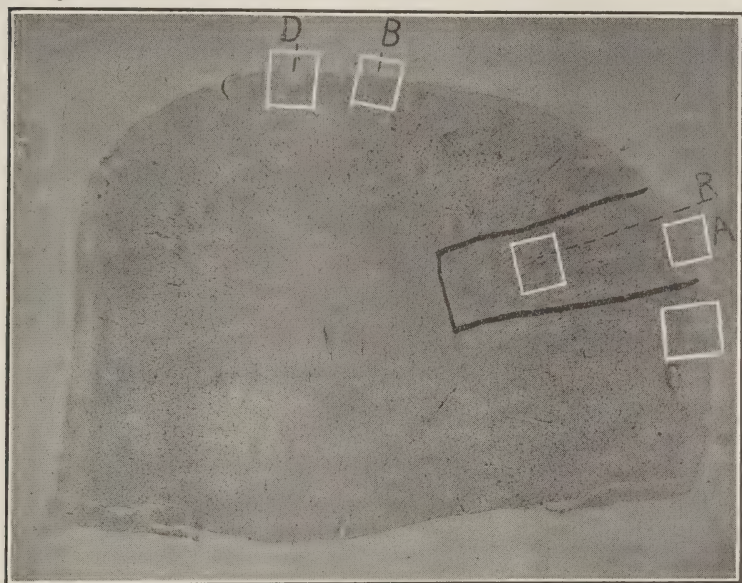


Plate XI, Fig. 32.

tion was taken is indicated by the letter D in Plate X, Fig. 29. The wide separation of the convolutions from one another, with resulting gaping sulci, is especially marked in this region. As previously mentioned, this is largely due to mechanical agencies, a central support permitting the opposite poles, owing to their weight, to become depressed and force open the sulci in this region during the process of hardening or fixation. In the removal of the pia mater, small portions of the adhering cortex have been removed in places. This is seen just below the letter O, and in many places in the convolutions both anterior and posterior to this. By referring back to Plate III, Fig. 7 of the normal brain

it will be seen that this section was taken from approximately the same relative position of the convolution as that seen in Plate III, Fig. 7, and, furthermore, the two convolutions are quite similar in shape; this convolution being somewhat broader and less rounded at the vertex than that of the normal brain, however. The outer cortical layer is here more irregular and torn than in Plate III, Fig. 7, and at the vertex of the convolution at *b* the outer part of the first layer is seen stripped off for some distance. This, of course, is an artefact, the detached part being more adherent to the stripped off pia than to the cortex below. This outer layer furthermore presents a somewhat irregular contour, being broken in places as at *c* and *d*. The layer is thicker opposite *a*, and on the left lateral aspect, than elsewhere. The second or pyramidal cell layer shows the general radial direction of the cells, but not so well marked as in Plate III, Fig. 7. The chief point of interest, however, is almost complete disappearance of the Betz cells, which are seen so prominently in this region in the normal brain in plates of this magnification (14 diameters). In photomicrographs of 100 diameters we will find this disappearance is not complete, and that the cells, although much atrophied and pale, so as to scarcely appear under the lower power, are much more apparent here, and are seen in all stages of dissolution. In this plate only scattered Betz cells are seen singly or in small groups at wide intervals, and these few are small, shrunken and, for the most part, pale. Fig. R is a drawing of the large Betz cell marked R in this plate. The nucleus is quite centrally situated, with a large distinct nucleolus and some faint chromatic substance. Some few chromophilic bodies are seen interspersed among the finely granular chromatic substance. The cell processes are pale, irregular, and soon terminate at a short distance from the cell-body. No pigment is found in this cell. Figs. S and T show two other near-by cells, the former containing an eccentric nucleus, irregular, shrunken cell-body, with some pigment at the left of the nucleus. The processes being irregular and short. The cell in Fig. T is much shrunken and distorted, the nucleus irregular, a small nucleolus, and but little diffuse chromatic substance. There are no basal processes, and the apical process is narrow and irregular in direction. Numerous capillaries are seen with thickened walls, and some with perivascular spaces. The third layer is indistinct, and fades into the white medullary substance, where many large capillaries with thickened walls, tortuous course and perivascular spaces are found. At *a*, and enclosed in ink lines, is the segment of this section, corresponding in position to that seen in Plate XI, Fig. 33. This section is 8 mm. wide, 6.5 mm. long, and 10 microns in thickness, fixed in 95 per cent alcohol, stained with methylene violet, and

other technique similar to all the other sections. Plate XI, Fig. 33, is the photomicrograph magnified 100 diameters of a segment corresponding to *a* of Plate XI, Fig. 32, and from a section taken from the same block, and but a short distance from the above. At this point the first or outer layer measures .25 mm. in thickness, whereas in Plate XI, Fig. 32, it would measure at least .40 mm. in thickness. It contains numerous neuroglia cells, with here and there a small pale nerve cell in which is situated a small nucleus surrounded by a mere trace of chromatic substance. Below this is the second or pyramidal cell layer, measuring 1.30 mm. in thickness. In the upper parts the pyramids are seen to be small and quite closely packed together. About the middle of the layer they are seen to be much larger and more scattered. Below this again, at *a*, is seen a region containing for the most part only small pyramids, and this finally merges into the lower portion of this layer, where are seen the large and giant pyramidal cells. These latter, or Betz cells, are seen in irregular groups or "nests," consisting of from one to several (six or more) cells. All of these cells show a diminished amount of chromophilic substance and no distinct granules. Various stages of advanced chromatolysis are seen, from a general diffusion of the chromatic substance to almost complete absence of the same. The cells are for the most part shrunken, the nuclei small and indistinct, displaced in many cases, and the cell processes atrophied and tortuous. Many of these larger cells contain a varying amount of yellowish pigment. Fig. U shows the cell marked U in the plate. No processes are seen, the cell-body is shrunken and contains but a small amount of diffused chromatin, showing no structure and pale in color. The nucleus is small, shrunken and indistinct, with a pale nucleolus. No pigment is to be seen in the cell. To the left of the cell-body is a small pericellular space. Several neuroglia cells are seen in apparent direct contact with the cell-body and others in the pericellular space and the wall of the latter. The cell just below and to the left of this in the plate is similar in regard to the amount of chromatin, is pale, and the nucleus and nucleolus are not visible. Three processes are seen given off at the base, but atrophied and extending for only a short distance. A small pericellular space is seen about this cell also. The cell just below and to the right of this, and marked V in the plate and seen in Fig. V, shows the same grade of chromatolysis. The remnant of a shrunken and atrophied process is seen at the base on the right. The shrunken nucleus, with pale nucleolus, is centrally situated, and below this are found diffused pigmentary deposits. Throughout the cell-body, less in the upper than in the lower part, is scattered pale diffuse chromatic substance, and without any definite arrangement. A narrow pericellular space is seen along

the right side of this cell. The general contour and lack of structure are seen in Fig. V. Here, too, neuroglia cells are seen lying upon and in close juxtaposition to the cell-body. Numerous blood vessels with thickened and tortuous walls and perivascular spaces are seen throughout this layer. The third or spindle or irregular cell layer here measures .90 mm. in thickness, and is made up of shrunk spindle and irregular cells, pale, and containing but a small amount of diffuse chromophilic substance. Many of them are surrounded by pericellular spaces of varying size. The letter

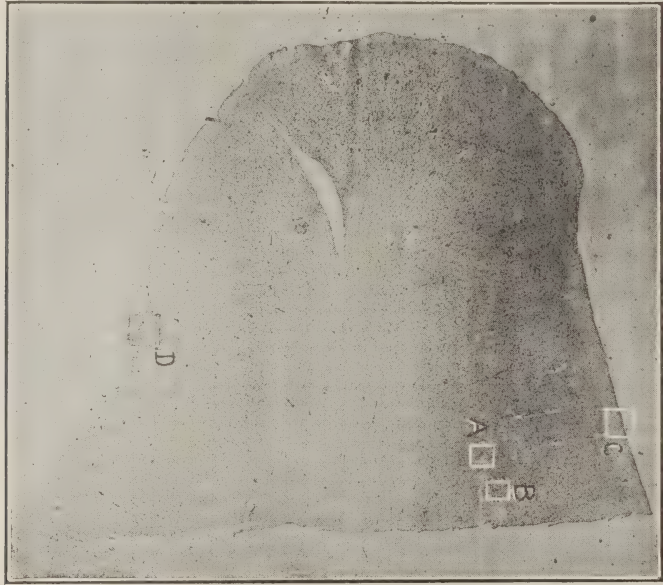


Plate XI, Fig. 34.

W indicates one of these spindle cells seen in the drawing, Fig. W. The polar processes are here filamentous, and distorted in direction, the cell-body shrunk and containing but a small amount of diffuse chromatic substance, so that it is pale in color, the nucleus is small and indistinct, and contains a small nucleolus. The cell-body is partially surrounded by a distinct pericellular space, with a neuroglia cell upon the edge at one point. Numerous blood vessels with thickened walls and perivascular spaces are also seen in this layer. The entire depth of the cortex here is 3.05 mm., all three layers being slightly thicker than in Plate III, Fig. 8, of Brain A, the first layer being .05 mm., the second .15

mm., and the third .20 mm., thicker than in the latter. As variations of this extent are found in sections of convolutions in close proximity, and even in different parts of the same section in any brain, it seems doubtful to the writer whether we can attach any special significance to such a slight variation, due possibly to mechanical causes, acting in embryonic or even post embryonic life. Counts of the nerve and neuroglia cells here show a decided fall-

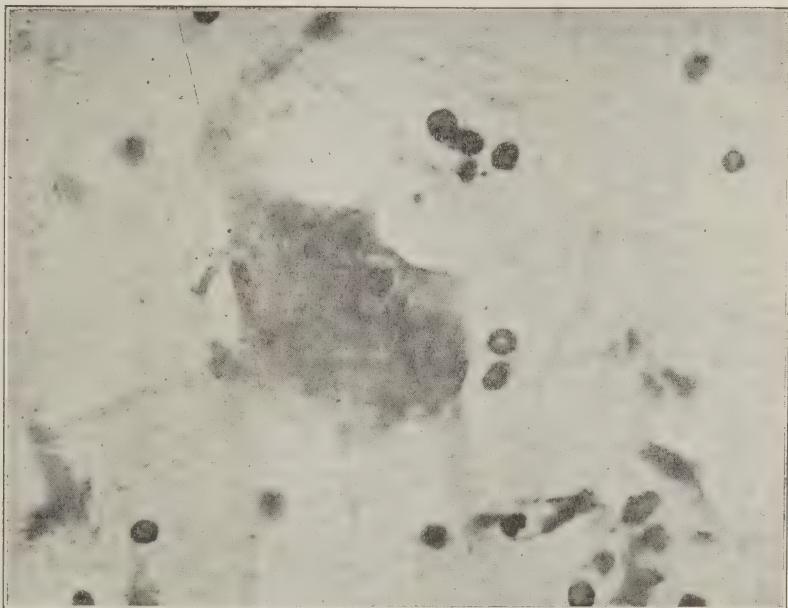


Plate XII, Fig. 35.

ing off in number of the former. This section is 6 2-3 microns in thickness and stained with methylene violet. The same method with the ocular net-micrometer was used, counting the nerve cells in eight different fields of thirty-six square millimeters, each in various parts of the second and third cortical layers, and the neuroglia cells in the same number of different fields in all these cortical layers. The average number of nerve cells to each square millimeter of surface of the section was found to be but 39.60, whereas the average number of neuroglia cells to the same area was found to be 204. The number of nerve cells is thus seen to be much diminished as compared to Plate III, Fig. 8, the corresponding region of the normal brain A, where there are an aver-

age of 95 nerve cells to the square millimeter of surface of the section. The number of neuroglia cells is also less here, being 204 in this plate in comparison to 293 in Plate III, Fig. 8. The latter section is, however, 10 microns in thickness, as compared to 6 2-3 microns for this plate, and as previously noted, owing to the small size and close aggregation of the neuroglia cells, more would be normally found in the thicker section, although this

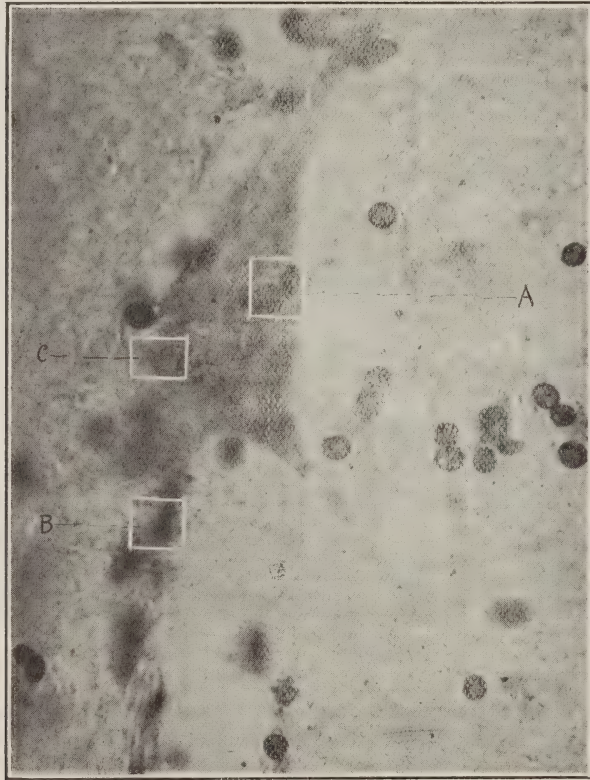


Plate XII, Fig. 36.

hardly accounts for the great difference, as both were stained in the same manner with methylene violet. Plate XI, Fig. 34, is a photomicrograph, magnified 14 diameters, from a section taken from the point L of the upper portion of the anterior central convolution of Brain B (see Plate X, Fig. 29), and as seen by referring to this latter, is from a more inferior portion of this gyrus

than the section represented in Plate XI, Fig. 32. This section was fixed in 95 per cent alcohol, is 6 2-3 microns in thickness, and stained with methylene violet. The first layer is seen to be fairly uniform in thickness at the lower two-thirds, as seen at *c*. Above, and at the vertex, and also along the anterior aspect of the convolution, as at *d*, the surface is more or less torn and irregular, due to carrying away of small fragments with the adherent pia. By referring to Plates XI, Fig. 32, and XIII, Fig. 39, the variations in shape and mechanical arrangement of the convolution in adjacent portions will be noted. In Plate XI, Fig. 32, the vertex is broadly rounded and somewhat flattened. In Plate XIII, Fig. 39, the vertex is more narrow and pointed, while in this plate, intermediate in position between the above two plates the vertex is less pointed. Large perivascular spaces are noted in many places throughout this section, especially marked in the upper portion to the left of the vertex, where but a fragment of the blood vessel is to be seen only at the lowest point of an enormous perivascular space. The radial arrangement of the cells is but poorly shown, the Betz cells seem to have disappeared almost entirely in the second layer, in marked contrast to the plates of the same magnification of Brain A. (Plates III, Fig. 7; IV, Fig. 9, and IV, Fig. 11.) Under a high magnification, however, the remnants of many of these Betz cells can be made out, showing various pathological changes. The other nerve cells also show a wealth of pathological change of varying degree and kind to which the nerve cell is subjected—all grades of chromatolysis, pigmentation, atrophy, and shrinkage of the nerve cells and processes, with larger or smaller pericellular spaces. Numerous capillaries, with thickened walls and perivascular spaces, are seen scattered throughout the various parts of the section. The actual size of this section is 8 mm. wide at the point opposite *d*, 8.5 mm. long, and 6 2-3 microns in thickness. At the points *a* and *b* are seen the Betz cells, shown under a magnification of 1,400 diameters in Plates XII, Fig. 35, and XII, Fig. 36. The former of these is a photomicrograph as stated, under a magnification of 1,400 diameters of the nerve cell, indicated by the letter *a* in Plate XI, Fig. 34. By referring to this latter plate, and then to Plate X, Fig. 29, we can locate almost the exact position of this cell in the cortex of Brain B. The cell-body is seen shrunken and deformed, lying in a pericellular space partially surrounding it. The nucleus is eccentric, being crowded over to the extreme edge of the cell-body on the right side. It is also shrunken and indistinct, but contains a well-marked and prominent nucleolus. The apical process above and to the right appears quite sharply deflected at a point but a short distance from the base, but this is not really the case, as the portion from *a* to the edge of the plate

is one of the walls of a blood vessel, the opposite wall not being seen in this plate at all. At the point *a*, this apical dendritic process appears to come in direct contact with this portion of the wall of the blood vessel, and is there lost to view. The other dendritic processes—seven in all—are shrunken, and contain a small amount of diffuse chromatin. Within the cell-body the chromatin is considerable in amount, and quite generally diffused in fine granules throughout the cell-body, lacking any definite structural arrangement. There is a somewhat greater amount at the base to the left and below the nucleus, and also at the upper part of the cell-body. Above and to the left of the nucleus, where the cell-body appears most pale, and extending into the base of the dendritic processes here, is a considerable mass of yellowish pigment. Numerous neuroglia cells are seen in the vicinity of this Betz cell. Plate XII, Fig. 36, is also a photomicrograph, magnified 1,400 diameters, of another of these Betz cells from the point *b* of Plate XI, Fig. 34. Here there is but little diffuse chromatin, confined principally to the base of the dendritic process given off on the right. All the lower part of the cell-body, excepting this portion, contains palely yellow pigment. The nucleus is displaced almost to the extreme edge of the cell-body, and lies just below the two neuroglia cells seen at *a*. The nucleolus, on this account, can not be determined at this plane. On microscopic examination the nucleus is found to be shrunken and indistinct. Numerous neuroglia cells are seen upon and in the immediate vicinity of this cell. The basal dendritic process *b*, which is but faintly seen here, owing to its lying in a somewhat lower plane, has eight neuroglia cells in close apposition to it. The dendritic process on the right can be traced for some distance, is somewhat shrunken, and contains no chromatic substance. Another small dendritic process is given off from the base opposite the point *c*, and being in a lower plane, only its base is seen here. It is colorless, and extends but a short distance from the cell-body. The apical dendritic process contains no chromatin, and as seen, has several neuroglia cells surrounding it a short distance from its base. A large pericellular space almost entirely surrounds the cell, excepting at the base to the left. Plates XII, Fig. 37, and XII, Fig. 38, are photomicrographs of the same magnification (1,400 diameters), taken from an adjacent section of the same block as the two preceding plates, and are also 6 2-3 microns in thickness. This section was prepared in exactly the same way, with the exception that methylene blue was used as the stain instead of methylene violet. This will be at once apparent upon noticing the neuroglia cells, which are here pale and washed out, many to the point of complete decolorization, so as not to be seen at all. The Betz cell in the center of Plate XII, Fig. 37, is seen surrounded by a large

pericellular space. All the processes, four in number, including the apical dendritic process, are pale, and terminate but a short distance from the cell-body. The nucleus is eccentrically situated near the wall at the left, is small, and contains a large nucleolus, but no nuclear network. Within the cell-body finely granular chromatic substance is found at the base below and to the right of

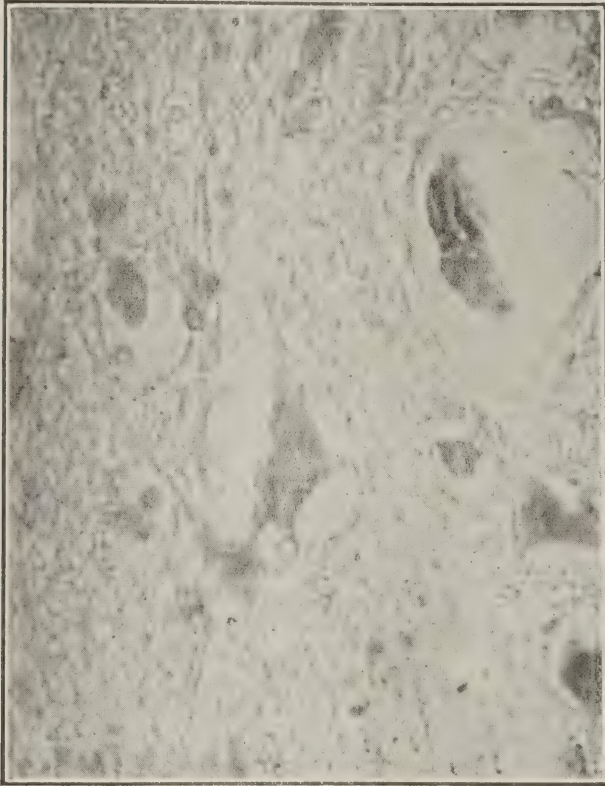


Plate XII, Fig. 37.

the nucleus. There is no pigment present. This cell presents an advanced stage of partial chromatolysis. Above and to the right is an almost indeterminable mass, which, under the 1-12 inch oil-immersion objective, appears to be the remnant of a capillary, surrounded by a large perivascular space. Other cells are seen here in various stages of disintegration, surrounded by large pericellular spaces, some containing none or only the remnants of den-

dritic processes, indistinct and shrunken nuclei, some distinct nucleoli and others none at all, and all in a more or less advanced state of chromatolysis, with but little of the diffused stained chromatic substance present. The neuroglia cells that are visible at all are pale and poorly stained, while many are almost or com-



Plate XII, Fig. 38.

pletely decolorized. Plate XII, Fig. 38, is a photomicrograph of a Betz cell on the same section and but a short distance from the cells of the preceding plate, and under the same magnification (1,400 diameters). Here there is almost complete chromatolysis, there being but little finely granular, diffuse chromatic substance scattered in the cell-body, slightly more at the base than above the nucleus. The latter is small, rounded, centrally situated, and

contains a large, rounded, distinct nucleolus. The three basal processes are narrow, pale, and terminate at no great distance from the cell-body. The apical process is pale and slender, extending for some little distance before coming to an end. Part of a pale, atrophied cell is seen to the right of the same. The ghosts of several neuroglia cells may be seen here, but most of them are completely decolorized.

Plate XIII, Fig. 39, as will be seen by referring to Plate X, Fig. 29, is from the lower portion of the upper third of the anterior central convolution, from the point marked O, and is magnified 14 diameters. This section, as already mentioned, is found to



Plate XIII, Fig. 40.

vary somewhat in shape from that of the preceding sections shown in Plate XI, Fig. 32, and Plate XI, Fig. 34, being narrower and more pointed at the vertex. From the strip marked *a*, and enclosed in ink, the structure and arrangement of the nerve cells were carefully studied, and both nerve and neuroglia cell counts made and will be referred to again later. The irregularity and difference in thickness of the first layer in different parts is to be noted, being especially thick at *b*, where it almost presents the appearance of an artefact, but under a high power no derangement of the structure can be made out. At *a* the layer is seen to

be very thin, owing to some of the surface of the cortex having remained adherent to and been stripped off with the thickened pia mater. Large perivascular spaces are to be especially noticed in the subcortical portion of the section. Here is to be noted the almost entire absence of the Betz or giant pyramidal cells in the lower part of the second layer, although some large pyramidal cells are to be found, they are not, however, so large nor are they arranged in such distinct groups as in the region higher up. Under higher magnification the remnants of some of these Betz cells are seen in various stages of necrosis and disintegration. The cortex lacks the distinct striated appearance seen in the sections of the normal cortex. This layer passes indistinctly into the third or spindle cell layer, which in turn is lost in the white medullary substance below. Numerous capillaries are seen, many with thickened walls and perivascular spaces throughout the section. This section was fixed in 95 per cent alcohol, sectioned 10 microns in thickness, stained with methylene violet, with other technique similar to that of all the previous sections. The actual size of the section is 9 mm. at its greatest width and 13 microns in length. The strip *a* varies from that of Plate XI, Fig. 33, only in the number and arrangement of the cells in the second layer. At this point the three layers are respectively .20 mm., 2.00 mm., and .90 mm. in thickness. The first layer contains some few scattered irregular nerve cells and numerous neuroglia cells. The second layer contains small pyramidal cells above, increasing in size until a little above the middle of the layer is a strip some .30 mm. in width, in which are to be seen large pyramidal cells arranged singly or in irregular groups. Some of these cells almost approach the giant pyramidal cells in size. Many of them show but a slight shrinkage and contain an almost normal amount of chromatic substance. The chromophilic bodies, however, are not as numerous or as large as in the normal brain. Other cells show a complete absence of chromophilic bodies, but contain the chromatic substance diffused throughout the cell in minute particles. Still others are pale, and contain but little chromatic substance. The nuclei are larger, less eccentric, and there is a less degree of atrophy of the cell-body and processes. Below this is a region of small pyramidal cells with only a few scattered larger pyramidal cells merging into the lower part of the layer, where large pyramidal cells are again seen scattered irregularly among the smaller cells. These large pyramidal cells are somewhat smaller than the giant pyramidal or Betz cells seen in the upper part of this convolution. These cells show more advanced pathological changes, in some cases marked atrophy and shrinkage with large pericellular spaces, atrophy of the dendritic processes, eccentricity and shrinkage of the nucleus, pigmentary deposits, complete absence

of or but a small amount of diffuse chromatic substance. The third layer presents the same general appearance as in the preceding plate (XI, Fig. 33), with many cells much shrunk and atrophied and little or no chromatic substance within the same, and surrounded by pericellular spaces of varying size. Numerous large and small capillaries are seen scattered throughout this



Plate XIII, Fig. 39.

section, with thickened walls and surrounded by larger or smaller perivascular spaces. The entire depth of the cortex here is 3.10 mm., the second layer being slightly thicker than in Plate XI, Fig. 33. The nerve cells were found to average but 36.50 to the square millimeter of surface of the section here; less than in any of the other plates, whereas the neuroglia cells average 289 to the square millimeter of surface of the section, almost as great a num-

ber as in the previous sections of the same thickness, ten microns, and stained with methylene violet. The most conspicuous difference between this and Plate XI, Fig. 33, higher up in the convolution, is the arrangement of the large pyramidal cells, the absence of the typical Betz cells, and on the whole less advanced pathological changes. Plate XIII, Fig. 40, is a photomicrograph magnified fourteen diameters of a section from the block B of Plate X, Fig. 29, and is thus seen to be situated a little above the middle of the posterior central convolution. This section is 6-2-3 microns in thickness, 8 mm. in length, and 9 mm. in width at the widest point, was fixed in 95% alcohol, stained with methylene blue, and other technique similar to that of all previously described sections. This section at the vertex, especially to the left, shows the striated appearance of cell arrangement very well, but upon the anterior and posterior aspects of the gyrus it is but indistinctly shown. The first layer is fairly uniform in thickness with broken spaces here and there, especially about the entrance of capillaries. This layer is somewhat thicker in certain places than in others. At *a*, for instance, it is thicker than at the vertex. This section has a broad, flattened vertex with considerable cortical area on both the anterior and posterior surfaces. The cortex opposite the angles *c* and *b* is somewhat thicker than elsewhere. This mechanical arrangement admits of a large, broad mass of fibres from various parts of this and the adjacent regions of the brain, leaving from and entering into relations with the cells of this portion of the cortex. The section is broken (an artefact) at the left and numerous capillaries are found scattered throughout all the cortical layers and also the white medullary center. The segment represented in Plate XIII, Fig. 41, was taken from a point corresponding to *a*, but from an adjacent section of the same block, and is magnified 100 diameters. It is from relatively the same position as the segment shown in Plate VII, Fig. 19, the strip *a* of Plate VI, Fig. 18, of the normal brain, but, as will be seen by referring to the latter, the shapes of the two sections are quite different. The distribution and arrangement of the cells in this plate is somewhat different from that in Plate VII, Fig. 19, also. In this latter, as already described, some large pyramidal cells are irregularly distributed in parts of the upper half of the second layer, followed by a narrow region made up almost exclusively of small pyramidal cells to be followed by larger pyramidal cells in the lower portion of this layer. In this plate, however, there are but few of the larger pyramidal cells in the upper portion of the second layer, there being practically only small pyramidal cells in this upper portion with the larger pyramids mostly in the lower portion of this second layer. The cell seen in the lower part near the center (*a*, Plate XIII, Fig. 41), approaches in size to the Betz cell type. The first or superficial

layer here contains nothing but capillaries and neuroglia cells and is .25 mm. in thickness. The second or pyramidal cell layer is 1.40 mm. in thickness and shows not only numerous capillaries with irregular and thickened walls, but also the cells in various pathological conditions. Chromatolysis is complete in many of these cells as in the large pyramidal cell marked *a* for instance, where only traces of minute finely powdered chromatin can be seen in some parts of the cell-body. Here also the nucleus is indistinct, with a well marked nucleolus, and the basal processes are much atrophied, terminating but a short distance from the cell-body. A small pericellular space is seen at the base of this cell-body. Many cells show eccentricity of the cell nucleus, shrinkage of the cell-body, various grades of chromatolysis, pigmentation, atrophy, and distortion of the dendritic processes and are surrounded by pericellular spaces. The capillaries are numerous, have thickened and irregular walls, and are surrounded by perivascular spaces of varying size and extent. Numerous neuroglia cells are also interspersed about the cells and capillaries in this layer. The third or spindle cell layer is only partially seen in this plate, and is about .90 mm. in thickness (only upper portion shown in Plate XIII, Fig. 41), and is similar to that in preceding plates of the motor region, and contains for the most part shrunken and irregular spindle and polygonal shaped cells, with numerous neuroglia cells and capillaries interspersed among them. Most of these cells show the various pathological processes mentioned above for the pyramidal cells. The cortex here thus measures 2.55 mm. in thickness, almost the same as for the two upper layers in Plate VII, Fig. 19, but here the third layer is .90 mm. in thickness as compared to .60 mm. in the above mentioned plate of the normal brain. Nerve and neuroglia cell counts were made here with the result that an average of 59.91 nerve cells and 134.37 neuroglia cells were found to the square millimeter of surface of the section as compared to an average of 185.50 nerve cells and 109.20 neuroglia cells for the same area in the strip of cortex represented in Plate VII, Fig. 19, of the corresponding region of the normal brain. Both sections were 6-2-3 microns in thickness and both stained with methylene blue. The difference in nerve cells is very marked, whereas the difference in number of neuroglia cells is not so great, and as the technique was identical throughout in the preparation of the two sections, the marked pathological process shown in this latter, it seems to the writer must be attributed as the cause of this difference to a large extent at least.

Parietal Region.—Plate XIV, Fig. 42, is a photomicrograph magnified fourteen diameters, of the parietal region from the point H. in Plate X, Fig. 29, this being a part of the supra-angular gyrus and from the same relative part of the gyrus as the

corresponding plate (VII, Fig. 20) of the normal brain. The section was fixed in 95% alcohol, stained with methylene violet, and treated otherwise as the preceding plates. This section measures 6 mm. in width, 16 mm. in length, and is 6-2-3 microns in thickness. Its shape is long and narrow and quite different from that of Plate VII, Fig. 20. At the vertex of the convolution

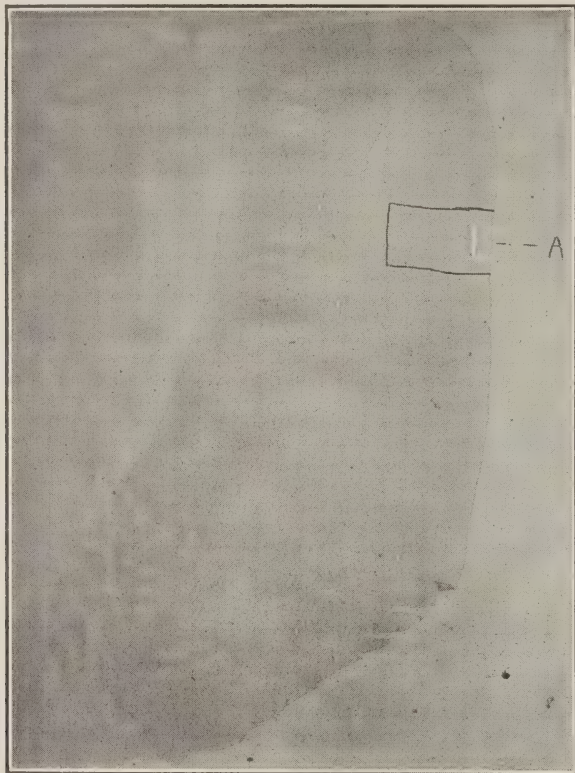


Plate XIV, Fig. 42.

the first layer has been cut away with the paraffin in preparing the block for sectioning serially with a Minot microtome, and, of course, this condition is an artefact. The first layer is seen to be fairly uniform in thickness with this exception. In the second layer it is difficult to determine the well marked striated arrangement of the cells, as seen in Plate VII, Fig. 20. Numerous perivascular and pericellular spaces are seen with or without the capil-

laries or cells, as the case may be. The third layer is indistinct and fades away into the white medullary center which also contains many pericellular and perivascular spaces. No doubt the fixation in 95% alcohol has produced some shrinkage, but by no means all, as the plates of the occipital convolution just posterior to this show a much less degree of shrinkage than is here present and not only every block taken from this brain was fixed in exactly the same way, but also some of the blocks from Brain A. At *a* and surrounded by ink lines is the segment of the section shown under a magnification of 100 diameters in Plate XIV, Fig. 43, and as is here seen is taken from the lateral aspect near the vertex, instead of from the vertex as in the corresponding plate of the normal cortex (Plate VII, Fig. 21). The first layer averages .25 mm. in thickness and contains numerous capillaries with thickened walls, and tortuous course with larger and smaller perivascular spaces. Numerous neuroglia cells are scattered throughout this layer. The second layer is here 1.50 mm. in thickness. Here, too, are seen numerous capillaries with thickened walls, irregular course, and large perivascular spaces. In the upper half of the layer the small pyramidal cells gradually are intermingled with larger pyramidal cells as the lower portions are reached, until just above *a* the largest pyramids of the section are seen singly or in groups in a comparatively narrow zone. Below and opposite *a* is a narrow zone, some .25 mm. in width in which there are practically no large pyramidal cells. Below this again is a very narrow zone at the lower part of this layer in which some few large pyramidal cells are seen scattered among the smaller and irregular cells. These cells present all stages of chromatolysis, pigmentation, shrinkage of the cell-body and processes and irregularity in direction of the latter, eccentricity and shrinkage of the nucleus and larger and smaller pericellular spaces. Numerous neuroglia cells are scattered throughout the layer. The third, or spindle cell layer, measures scarce .50 mm. in thickness and contains for the most part shrunken and irregular spindle cells in larger or smaller pericellular spaces, in many instances in various stages of chromatolysis. Numerous neuroglia cells in many cases appearing in direct contact with the cell-body are seen in this layer, as well as in the above layer. The vascular changes are also similar. The entire depth of the cortex here is thus 2.25 mm., somewhat less than in Plate VII, Fig. 21, but about the same as on the lateral aspect of this latter plate. Nerve and neuroglia cell counts were made here in the same manner as in previous sections, and it was found that there was an average of 68.63 nerve cells and 192.70 neuroglia cells to each square millimeter of surface of the section as compared to 104 nerve cells and 180.90 neuroglia cells in the corresponding region of Brain A,

both sections being of the same thickness and stained in the same manner with methylene violet.

Temporal Region.—Turning to the region below this we come to the temporal region represented here by a section from the first temporal convolution near its anterior extremity, from the

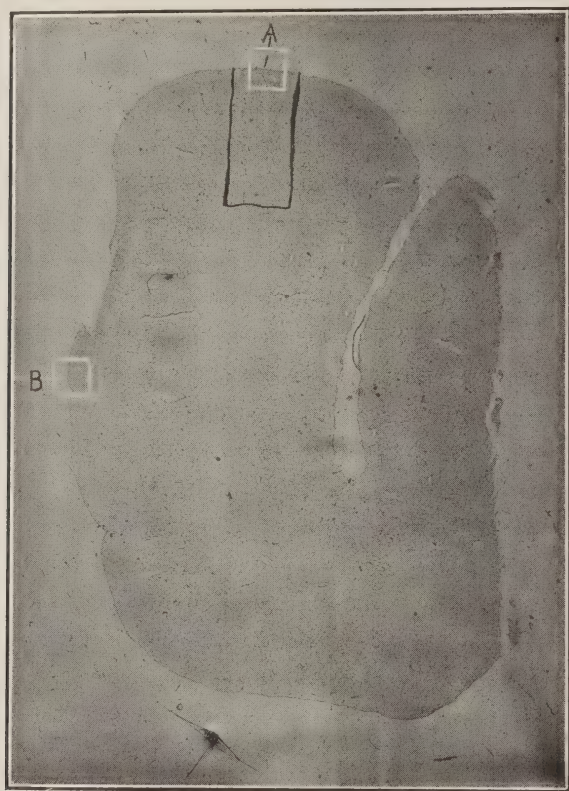


Plate XIV, Fig. 44.

point *I* in Plate X. Fig. 29, and shown under a magnification of fourteen diameters in Plate XIV. Fig. 44. This convolution is also much narrower than the corresponding convolution of *Brain A*, seen in Plate VIII, Fig. 22, thus having a somewhat dissimilar mechanical arrangement. To the right is seen the first temporal fissure and a portion of the second temporal convolution. The convolution measures 5.5 mm. in width and 8 mm. in depth from

the vertex to the point on the level with the bottom of the first temporal fissure and is 6 2-3 microns in thickness. The convolution at this point is seen on the right to curve around and fuse with the second temporal convolution, being free above at *b*, which is the lower boundary of the fissure of Sylvius at the beginning of the posterior branch. At *a* and enclosed in ink lines is the segment of the section shown in the following plate under a magnification of 100 diameters. The first or superficial layer is quite uniform in thickness, broken in several places, especially above *b* on the superior surface. The second layer shows the striated arrangement of the cells only very indistinctly at some few points and contains many pericellular and perivascular spaces, some with and others without contents. The third layer is indistinct and fades into the white medullary center which is narrow and passes inferiorly over to connect on the right with that of the second temporal convolution.

In Plate XV, Fig. 45, strip *a* of Plate XIV, Fig. 44, under a magnification of 100 diameters, the pathological conditions are seen to be well marked. The section is 6 2-3 microns in thickness and stained with methylene violet. Here the first layer averages about .20 mm. in thickness and to the right a fragment of a blood vessel is seen at the surface, but penetrating the cortex in a different plane. In the center and reaching into the second layer is a large capillary with thickened walls, with larger caliber above and smaller below, being somewhat funnel shaped and surrounded by a large perivascular space. Some smaller fragments of capillaries and neuroglia cells are scattered about in the layer. The second or pyramidal cell layer is 1.50 mm. in thickness and presents very much the same general plan of arrangement as the corresponding region in the normal brain, as seen in Plate VIII, Fig. 23, and also in the region of the first frontal convolution, as seen in Plate II, Fig. 3, and XI, Fig. 31. Small pyramidal cells almost exclusively are seen in the upper part of the layer below this gradually increasing in size to the middle of the layer. Then opposite *a* is a narrow strip some .20 mm. in thickness in which there are small pyramidal cells almost exclusively. Below this again and in the lower part of this layer are larger pyramidal cells, intermingled with smaller pyramidal and irregular shaped cells. Here, too, the majority of the cells are seen shrunken and surrounded by larger and smaller pericellular spaces. Various stages of chromatolysis, shrinkage and eccentricity of the nucleus and atrophy of the cell processes are to be observed. The blood vessels show the same pathological conditions as in the upper layer and numerous neuroglia cells are interspersed thickly everywhere.

The third layer is .80 mm. in thickness and presents the same

pathological conditions of the cells and blood vessels as previously described for this layer in Plate XIV, Fig. 43, of the parietal region. The entire depth of the cortex here is 2.50 mm., somewhat thicker than the corresponding region of *Brain A*. Nerve and neuroglia cell counts were also made here and in a similar manner, an average of 56.74 nerve cells and 205.90 neuroglia

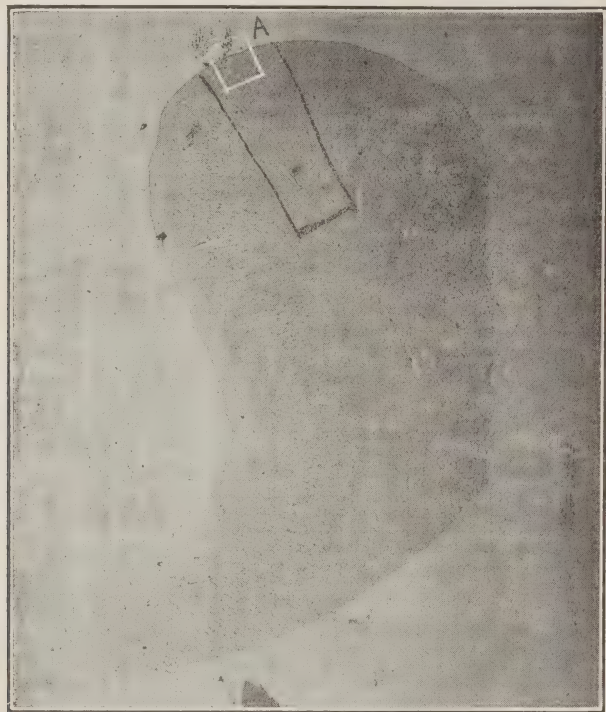


Plate XV, Fig. 46.

cells being found to the square millimeter of surface of the cortical portion of this section, as compared to 146. nerve cells and 131.90 neuroglia cells to the square millimeter of the surface of the cortex in the corresponding region of *Brain A*. In the latter methylene blue was the stain used, the thickness of the sections being the same in both cases, and as has been seen in every instance the methylene violet stain has greater affinity for the neuroglia cells than the methylene blue, whereas, the nerve cells stain about the same with the one as with the other.

Occipital Region.—Plate XV, Fig. 46, is a photomicrograph magnified fourteen diameters of a section taken from the block *J* of Plate X, Fig. 29, situated in the upper portion of the lateral aspect of the occipital convolution. The section is 5 mm. wide, 8 mm. in length and 6-3 microns in thickness, was fixed in 95% alcohol, and stained with methylene violet. As may be observed, upon the posterior aspect, the sulcus is very shallow, the cortex here fusing with that of the adjacent gyrus not far from the vertex, whereas anteriorly the sulcus is well marked and quite deep. The first or outer layer is thicker both on the posterior and anterior aspects than at the vertex and is quite uniformly regular in outline. The second layer is well marked and shows the striated arrangement of the cells quite as well as in *Brain A*, at this point. Numerous large cells, some of which quite approach the Betz cells in size and structure, are seen scattered in the middle and lower portions of the layer. The third or spindle cell layer below is gradually lost in the white medullary substance. Throughout the entire section the cells and their arrangement are well made out, the capillaries although having thickened walls and somewhat tortuous are less conspicuous, the pericellular spaces smaller and less numerous, and most of the cells are fairly normal and show no distinct pathological changes. At *a* and enclosed in ink lines is the position of the segment in the adjacent section from which Plate XV, Fig. 47, was taken. This latter corresponds most favorably with Plate IX, Fig. 25, from the corresponding region of the normal brain and some of the nerve cells here in Plate XV, Fig. 47, are larger and more numerous than in the corresponding section from *Brain A*. The first or outer layer is uniform in outline and measures here but .15 mm. in thickness. It contains numerous neuroglia cells and some scattered small nerve cells similar to those described in Plate II, Fig. 3, and seen in the text as Figs. C, D, E, and F. The second or pyramidal cell layer is of special interest and measures 1.40 mm. in thickness, being somewhat deeper than in Plate IX, Fig. 25, but with a similar arrangement of cells. Above are small pyramidal cells only, then larger pyramidal cells are seen in the deeper portions until about the middle of the layer is a region containing very large pyramidal cells, same as the one indicated by the letter X, for instance, being similar in size and structure to the Betz cells. Below this at *a* is a region .30 mm. in thickness in which numerous small pyramidal cells are alone seen. Below this again is a narrow zone containing some larger pyramidal cells scattered amongst the small pyramidal and irregular cells. The cell marked in the plate by the letter X is seen magnified in Fig. X. It is very large, contains irregularly elongated and linear chromophilic bodies of considerable size and arrangement in a

more or less parallel direction to the cell-body and extending into the dendritic processes. The nucleus is large, rounded, distinct, and centrally located with a distinct rounded nucleolus and indefinite chromophilic network within. No processes are given off at the base in this plane, but a large process is given off upon the right at the level of the nucleus and soon divides into two branches. Another process is given off to the left on the opposite side of the cell-body at a level just above the nucleus. The apical dendritic process contains numerous linear chromophilic bodies extending for some distance into the same. This is a stichochrome nerve cell of the somatochrome class, and, in size and structure, is similar to the Betz cell. The two cells to the extreme right are similar in structure but not as large as the above. There are no pathological changes to be determined in the cells of this layer. Several small capillaries are seen with somewhat thickened walls, indicating that possibly the blood vessel changes are the first to be found in the disease, but of course much more extended observation and study would be required to determine the full relation of all these pathological processes to one another. The third or spindle cell layer is here .90 mm. in thickness and made up of spindle and irregular polygonal cells, quite normal in appearance for the most part; some, however, showing slight shrinkage, pericellular spaces, and slight beginning chromatolysis. Some capillaries with thickened walls and perivascular spaces are to be observed here also. The entire thickness of the cortex measures 2.45 mm. being .45 mm. thicker than in Plate IX, Fig. 25. Nerve and neuroglia cell counts were made here with the results that 62.50 nerve cells and 215.01 neuroglia cells were found on an average to each square millimeter of surface of the cortex here, as compared to 164. nerve cells and 199. neuroglia cells in the same area of the corresponding section of *Brain A*, both sections being 6-2-3 microns in thickness and stained with methylene violet. From a study of the above plates from these two brains, the cortex in *Brain A*, would seem to be thinner and more compact, whereas, the cortex of *Brain B*, on an average measures somewhat thicker, yet the cells seem more scattered, so that a very much higher average of nerve cells is to be found in the former, far more it seems to the writer than could be accounted for by the possible destruction of some of the cells in the sections of *Brain B*, due to pathological agencies.

The careful detailed study of the sections from various parts of the different regions of *Brain A*, practically normal from a histological standpoint, and of corresponding sections of *Brain B*, a case of advanced dementia paralytica in the manner as described

in this article has led the writer to the following conclusions:

- I. Photomicrography is a useful and valuable adjunct to such work, giving greater accuracy in relations of parts to one another than is possible in drawings and showing conditions as they actually appear to the eye of the observer in each particular plane when the sections are seen under the microscope. Powers of 14 or 20 diameters show very well relations of the layers to one another, and also the relation of the groups of large cells; such as the Betz cells in the central regions, for instance. Powers of 100 diameters are well adapted to the study of the more minute detail of these relations of the layers and of various cells to one another, and also some of the morphology of the individual cells can be made out. Powers of 1,000 to 1,500 diameters show the internal structure of the cell to excellent advantage, but only in the very limited plane at which the object is focussed. On the other hand, the disadvantage of photomicrography is that everything is shown in any one plane so that special parts or structures are not brought out as prominently as in schematic drawings, and on this account all such work should be accompanied by such drawings to make it more complete. It is furthermore often quite difficult to obtain any one plane showing most of the cell structure, to say nothing of all of the same.

- II. Artefacts of many kinds are to be found in such work, and should be carefully avoided and eliminated in every instance before definite conclusions are drawn from any series of observations. Pressure and other mechanical causes acting before death, that due to manipulation at autopsy, artefacts resulting from fixation, imbedding, sectioning, staining, decolorizing, and even mounting, are all possibilities, and are often most difficult to determine in contradistinction to the pathological changes.

- III. The method of transporting material by means of small metallic boxes or similarly sized phials, permitting of a free circulation of various kinds of fixative fluids, without injury, and also permitting of subsequent exact localization, the process requiring a minimum of time and space is, to the mind of the writer, of decided practical value in such work. This method is fully described in the text under the heading of "Technique."

- IV. Nerve and neuroglia cell counts have been alluded to

above, but a few remarks upon method may be of value. The method of counting the same from photomicrographs of moderate magnification is not accurate, as cells may overlap or be arranged in dense groups or their images may not all be sufficiently distinct so as to differentiate them properly, especially does this apply to the neuroglia cells, which are very small and often closely aggregated. Photomicrographs magnified a thousand diameters or more, although showing all the cells visible, are much too limited in area. For these reasons the use of the ocular net-micrometer was found to be the best and most accurate for this work, using a stage micrometer to determine the relation of the ocular field to the actual size of the section. In this way the exact number of nerve and neuroglia cells can be determined for any number of fields and an average obtained for any area, as was done in various plates in the brains employed in this work, as seen in Table II. As will be noted by referring to this latter, there is considerable variability in the number of nerve and neuroglia cells not only in different cortical regions but also from adjacent sections of the same region. The stain is seen to play an important rôle here in the resulting average number of neuroglia cells, methylene violet having a greater affinity for these cells than methylene blue. Sections stained by the latter cannot always be depended upon to show all the neuroglia cells. In comparing the normal paretic brain there is marked diminution of the nerve cells in the latter as compared to the former, whereas the neuroglia cells, making due allowance for variability, are somewhat increased in number in the paretic brain.

V. The thickness of the layers is not constant in different parts of the same gyrus, nor even in different portions of a single transverse section of a gyrus, to say nothing of different regions of the cortex. In the same way there is greater or less variability in the shape and mechanical arrangement of each gyrus in contrast to the other gyri making up the surface of the hemisphere and different parts of the same gyrus vary in this respect. This will be best observed by referring to the several plates of both Brains A and B, magnified 14 diameters, and of the various regions of the cortex.

VI. After a quite extended perusal of the literature upon the

structure of the cortex as presented in the first portion of this article, the writer inclines to the view of a three-layered type of cell arrangement in the cortex, as the type with, of course, variations in special parts, as the cornu ammonis, for instance. This is shown in Table I, and brief is as follows: 1, or superficial layer, containing but few nerve cells, many neuroglia cells, and many chiefly tangential fibers. The first two elements only are seen with the Nissl stain, whereas the latter element is most conspicuous with Weigert's stain, and for this latter reason Ramon y Cajal designated it as the tangential fiber layer; 2, or pyramidal cell layer, to reduce the layering of the cortex to its lowest terms, and including the small, large, and giant or Betz pyramidal cells all in this one layer; 3, or spindle cell layer, including both the spindle cells which are in the majority and the less numerous irregular polygonal cells. Below these three layers is the sub-cortical white medullary substance.

VII. The study of the internal structure of the normal brain from a histological standpoint (Brain A) by the Nissl method shows four principal types of nerve cells: (1) The small rounded nerve cells of the first layer with or without one or more dendritic processes—rarely more than two, containing a rounded nucleus almost filling the body of the cell, this latter surrounded by a partial or complete narrow band of finely granular chromophilic substance. The nucleus contains a well marked nucleolus and slightly stained protoplasmic substance. These are co-called karyochrome nerve cells of Nissl's classification. (2) The pyramidal cells of the second layer, of which there are four varieties (a) the small pyramidal cells, (b) the large pyramidal cells, (c) cells intermediate in size and structure between the large pyramidal cells and the typical giant pyramidal or "Betz" cells, and (d) the giant pyramidal or "Betz" cells. In the smaller pyramidal cells it is difficult to distinguish distinct chromophilic granules, but the chromatin is arranged in larger or smaller finely granular masses in various parts of the cell-body, sometimes aggregated about the nucleus, at other times near the base or dendritic processes. The larger pyramidal cells, however, have the increasing amount of chromatic substance arranged in more or less distinct larger and smaller chromophilic granules, and these arranged in a direction

parallel to the surface of the cell-body. Still larger in size and more distinct in the arrangement of chromophilic granules in this general parallel manner are the intermediate variety of these pyramidal cells, which, although larger than the average pyramidal cell are considerably smaller than the typical Betz cell, and have a wider range of distribution not only in the vertical extent of the second layer of the cortex, but also in the different regions of the cortex, being found in the central, parietal, temporal, and occipital regions. Finally the largest in size and most distinct in structure, especially in the arrangement of the chromatic substance, are the giant pyramidal or Betz cells, in which larger size, large distinct nucleus and nucleolus, and also large parallel arranged chromophilic granules are seen in the cell-body, and often extending far up into the dendritic processes. These are localized, for the most part, in the lower portion of the second layer in certain areas of the central convolution and the posterior portion of the superior frontal convolutions. These pyramidal cells as a whole can best be designated as stichochrome nerve cells of the somatochrome class in Nissl's classification, although the smaller pyramidal cells may simulate more the gryochrome nerve cells of this same class, owing to the indistinct arrangement of the chromatic substance.

(3) The spindle cells found in the third layer and containing a very large nucleus with well marked nucleolus. The nucleus is often so large that it seems disproportionate to the size of the latter. The chromatic substance here is finely granular and arranged in irregular masses or heaps about the nucleus, and extending into the base of the dendritic processes, these latter, usually two in number and opposite, producing a bipolar condition. These cells are gryochrome nerve cells of the somatochrome class.

(4) Finally irregular or polygonal nerve cells are found in the second and third layers. These cells are irregular in shape, with three or more dendritic processes, a large nucleus containing a well marked nucleolus. In the cell-body are irregular finely granular masses of chromatic substance similar to the spindle cells in this respect, so could fall in the same classification as gryochrome nerve cells of the somatochrome class. In the cerebellum, the Purkinje cells, with their chromatic substance more or less arranged in a network, are classified as arkychrome nerve cells of

the somatochrome class, whereas the small nerve cells of the granular layer, with small nucleus, appear only partially surrounded by the cell-body, are classified as cytochrome nerve cells. The four types of cells above mentioned, as found in the cerebral cortex, were seen in all the regions of the external surface of the hemisphere studied. In addition to these cellular elements of the nervous tissue the neuroglia cells of the interstitial tissue were everywhere to be seen, and in the walls of the blood vessels the vascular cellular elements were also to be found.

VIII. Previous investigators, in the study of the cerebral cortex in dementia paralytica by the use of the Nissl method, have noted the following pathological changes: Various stages of cell degeneration up to complete destruction of the same, consisting of diminution, disintegration, and vacuolization of the cell protoplasm, all stages of chromatolysis up to complete disappearance of the chromatic substance, shrinkage with deformity of the contour of the cell-body, atrophy of the dendritic processes, various degrees of pigmentation and pigmentary deposits in the cell-body, also adjacent to blood vessels; shrinkage with diminution in size, irregularity, compression, vacuolization, and eccentricity of the nucleus with even extrusion of the same from the cell-body by the rupture of the cell wall, or complete sclerosis of the nucleus with homogeneous and tinged contents or crystalline deposits, nucleolus displaced to nuclear wall or indistinguishable or vacuolated, calcareous deposits in the form of fine granules, crumbs, plaques, or stalactitic masses intensely colored with methylene blue and found in the bodies of the sclerosed cells, part or whole of cell entirely bleached, complete necrosis of cells, reduction in number of nerve cells, thickening of pia with septa projecting into the cortex, granular crowding of variously stained granules, obscure layering of the cortex, multiplication of white corpuscles, proliferation of neuroglia cells, increase in and dilatation of capillaries and arterioles, with thickening of walls of same by encasement of latter with lymphatic corpuscles, and finally proliferation of interstitial network. The writer has observed all of these changes excepting the following: vacuolization of the cell protoplasm was not observed in the paretic material examined in this work, no pigmentary deposits were observed outside of the nerve cell bodies, al-

though varying amounts were found in many of the nerve cells. No vacuolization of the nucleus or extrusion of the same was found here, no crystalline or calcareous deposits were made out in any of the cells, no septa were observed penetrating from the thickened pia into the cortex, multiplication of white corpuscles was not observed, and proliferation of neuroglia cells as seen from the nerve and neuroglia cell counts as tabulated in Table II. seems to have existed to but a slight extent in small localized places, as about some of the necrosed nerve cells. The writer does not mean to say that this may not occur in some cases, for on the contrary he is inclined to think it may occur under certain conditions, either localized or more general. Also no proliferation of the interstitial network was observed here. About the nerve cells pericellular spaces of greater or less extent were observed in many cases. In some only a small portion of the cell-body, or a single dendritic process, was surrounded by a limited space; in many, however, a large portion of all the cell-body was thus surrounded. Perivascular spaces were also observed of varying size and extent about many of the blood vessels of the cortex. Pigmentation, in addition to being present in many of the nerve cells of the first and second layers of the cortex in the central regions, was also observed in cells of these layers in the frontal region as well; also in the parietal and temporal regions, but not in the occipital region. The pathological process here was most severe in the central and frontal regions, extending to a lesser extent, but still very marked, into the temporal and parietal regions, whereas the occipital region almost entirely escaped and appears practically normal. In the regions involved in this case the disease seems to be a chronic disease of the nerve cells with pigmentary degeneration and a necrosis of the cell-body, partial or complete, with accompanying involvement of the blood vessels. The relation of the vascular and cell changes to one another and the order of procedure in time is one that the writer believes requires much more extended investigation before it can be answered satisfactorily.

In conclusion the writer gratefully acknowledges the kindly interest, suggestions, and assistance offered by Professor H. Fairfield Osborn, Dr. Oliver S. Strong, and Dr. Edward Leeming, of Columbia University; Dr. Ira Van Giesen, former Director of the

Cajal 1890	Gowers 1893	
	Motor Type	Sensory Type
Common Type	Superficial	Superficial
Molecular	Superficial	Superficial
I Zone	I Layer	I Layer
Zone of Small Pyramidal II Cells	Small Pyramidal Cell II Layer	Small Pyramidal Cell II Layer
Zone of Large Pyramidal III Cells	Large Pyramidal	Large Pyramidal
	III Cell Layer	III Cell Layer
	Ganglion Cell IV Layer	Granular IV Layer
		Ganglion Cell V Layer
Zone of Polymorphous IV Cells	Fusiform Cell V Layer	Fusiform Cell VI Layer
White Central Substance	White Central Substance	

Gennari 1782	Vicq d'Azyr 1786	Meckel 1812	Baillarger 1840	Remak 1841	Kolliker 1852	Berlin 1858	Clarke 1863	Lays 1864	Arndt 1867	Meynert 1867	Cleland 1870	Henle 1870	Charcot 1871	Lewis 1876	Majör 1876	Krause 1876	Retz 1881	Golgi 1885	Schwalbe 1885	Obersteiner 1887	Cajal 1890	Gowers 1893	Hannaberg 1895	Edinger 1896	Starr 1896	Missl 1899	Lawrence 1902			
Common Type	Occipital Region	Common Type	Occipital Region	Common Type	Common Type	Common Type	Common Type	Common Type	Common Type	Common Type	Common Type	Common Type	Common Type	Motor Type	Sensory Type	Common Type	Common Type	Motor Type	Sensory Type	Common Type	Common Type	Motor Type	Sensory Type	Common Type	Common Type	Common Type	Common Type	Common Type		
Gray I Layer	External Gray I Layer	External Gray I Layer	External Gray I Layer	External White I Layer	Weisse I Rinderschicht	Weisse I Rinderschicht	First-I-Fiber Layer Second-II- Fiber Layer	External I Layer	First Tangential Fiber-I-Layer Second Tangential Fiber-II-Layer	Superficial I Layer	Superficial I Layer	Outer I Layer	Superficial I Layer	Superficial I Layer	Superficial I Layer	I Superficial Layer	Superficial I Layer	Neuroglia I Layer	Neuroglia I Layer	Superficial Part. I	Layer Free I	External I Layer	a Molecular I Zone	Superficial I Layer	Superficial I Layer	Superficial I Layer	Layer of Tangential I Fibers	Superficial I Layer	Layer in I Cells	1 Superficial I Layer
	Layer	Layer	Layer	External Gray II Layer				Layer of II										Small Pyramidal II Coll Layer	Small Pyramidal II Coll Layer	Superficial or Small I Pyramidal Cell Layer	from Colls I	Small Pyramidal II Cell Layer	c Zone	I Layer	I Layer	Small Pyramidal II Cell Layer	Layer of Small Pyramidal II Colls	Layer of Small Pyramidal II Colls	Layer of Small II Pyramids	2 Layer of Pyramidal II Cells
	White Layer or II Line of Gennari	White Layer or II Line of Vicq d'Azyr	White II Layer	Striae Externe or III Line of Baillarger	Grauer, oder Graueröthlicher II Schicht	II	Pale Cellular	Small Cells	Granular III Layer	Small Pyramidal II Cell Layer	Pyramidal Cell II Layer	Outer Spherical II Cell Layer	Small Pyramidal Cell II Layer	Small Pyramidal Cell II Layer	Small Pyramidal Cell II Layer	Small Pyramidal Cell II Layer	Layer of Small III Pyramids	Large Pyramidal Cell III	Granular III Layer	Pyramidal Cell Layer	d Zone of Small Pyramidal II Cells	Small Pyramidal II Cell Layer	Zone of Small Pyramidal II Cells	Small Pyramidal II Cell Layer	Small Pyramidal II Cell Layer	Large Pyramidal II Cell Layer	Layer of Small II Pyramids	3 Layer of Pyramidal II Cells		
	Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells
	Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells	Large Pyramidal Cell III	Large Pyramidal Cell III	Small Pyramidal and Irregular IV Coll Layer	Layer of Large Pyramidal III Cells	Layer of Small III Cells	4 Layer of Pyramidal II Cells	
Internal Gray III Layer	Gray I Layer	Internal Gray III Layer	Gray I Layer	Middle Gray IV Layer	Grauer, oder Substantia II Schicht	IV Medium	Transverse III Fiber Layer	Ganglion Cell IV Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Large Pyramidal Cell III Layer	Pyramids	III	Longitudinal Fiber IV Layer	Middle or Large Pyramidal II Cell Layer	Baillarger's Line or Grenz- schicht	Large Pyramidal Cell III	Zone of Large Pyramidal III Cells							

TABLE

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EXPLANATION OF PLATES.

Plate I. Fig. 1. Brain A. Left hemisphere. Electrocuted case, normal histologically.

indicated by the figure 1 in Plate I. Fig. 1. Section 6-2-3 microns in thickness stained with methylene blue and magnified 14 diameters.

Plate II. Fig. 3. Brain A. First frontal conv. Strip *a* of Plate I, Fig. 2, magnified 100 diameters.

Plate II. Fig. 4. Brain A. First frontal conv. Cell *G*. of Plate II, Fig. 3 magnified 1400 diameters.

Plate II. Fig. 5. Brain A. First frontal conv. Cell *H* of Plate II, Fig. 3 magnified 1,400 diameters.

Plate III. Fig. 6. Brain A. First frontal conv. Group of spindle cells *I* of third layer of cortex shown at *I* in Plate II, Fig. 3 magnified 1,400 diameters.

Plate III. Fig. 7. Brain A. Ant. cent. conv. Upper third, taken from point indicated by the figure 2 in Plate I, Fig. 1. Section 10 microns in thickness stained with methylene violet, and magnified 14 diameters.

Plate III. Fig. 8. Brain A. Ant. cent. conv. Upper third. Strip *a* of Plate III, Fig. 7 magnified 100 diameters.

Plate IV. Fig. 9. Brain A. Ant. cent. conv. Upper third. Section

from same block and adjacent to that shown in Plate III, Fig. 7. Section 10 microns in thickness, stained with methylene blue, and magnified 14 diameters.

Plate IV. Fig. 10. Brain A. Ant. cent. conv. Upper third. Strip *a* of Plate IV, Fig. 9, magnified 100 diameters.

Plate IV. Fig. 11. Brain A. Ant. cent. conv. Upper third. Section taken from same block and adjacent to that shown in Plate IV, Fig. 9. Section 10 microns in thickness, stained with methylene blue, and magnified 14 diameters.

Plate IV. Fig. 12. Ant. cent. conv. Upper third. Giant pyramidal cell from lower portion of second layer of cortex from section adjacent to that shown in Plate III, Fig. 7. Section 10 microns in thickness, stained with methylene violet and magnified 1,400 diameters.

Plate V. Fig. 13. Brain A. Ant. cent. conv. Upper third. Portion *a* of Plate IV, Fig. 11, magnified 825 diameters.

Plate V. Fig. 14. Brain A. Ant. cent. conv. Upper third. Two giant pyramidal cells from lower portion of second layer of cortex from section adjacent to that shown in Plate IV, Fig. 9. Section 10 microns in thickness, stained with methylene blue and magnified 1,400 diameters.

Plate V. Fig. 15. Giant pyramidal cell from Ant. cent. conv. Upper third, lower part of second layer from approximately the same region as the cells shown in Plate V, Fig. 14, and stained with methylene blue, Section 10 microns in thickness, and magnified 1,400 diameters.

Plate VI. Fig. 16. Brain A. Ant. cent. conv. Lower third, taken from point indicated by the figure 4 in Plate I, Fig. 1. Section 10 microns in thickness, stained with methylene violet, and magnified 10 diameters.

Plate VI. Fig. 17. Brain A. Ant. cent. conv. Lower third. Strip *a* of Plate VI, Fig. 16, magnified 100 diameters.

Plate VI. Fig. 18. Brain A. Post. cent. conv. Middle third. Section taken from the point indicated by the figure 5 in Plate I, Fig. 1. Section 6 2-3 microns in thickness, stained with methylene blue, and magnified 10 diameters.

Plate VII. Fig. 19. Brain A. Post. cent. conv. Middle third, upper portion. Strip *a* of Plate VI, Fig. 18, magnified 100 diameters showing first and second layers and upper portion only of third layer.

Plate VII. Fig. 20. Brain A. Supra-marginal conv. of parietal region taken from point indicated by the figure 7 in Plate I, Fig. 1. Section 6 2-3 microns in thickness, stained with methylene violet, and magnified 14 diameters.

Plate VII. Fig. 21. Brain A. Supra-marginal conv. of parietal region. Strip *a* of Plate VII, Fig. 20, magnified 100 diameters.

Plate VIII. Fig. 22. Brain A. First temp. conv. taken from point indicated by the figure 6 in Plate I, Fig. 1. Section 6 2-3 microns in thickness, stained with methylene blue, and magnified 14 diameters.

Plate VIII. Fig. 23. Brain A. First temp. conv. Strip *a* of Plate VIII, Fig. 22, magnified 100 diameters.

Plate VIII. Fig. 24. Brain A. Occipital conv. taken from point indicated by the figure 8 in Plate I, Fig. 1. Section 6 2-3 microns in thickness, stained with methylene violet and magnified 14 diameters.

Plate IX. Fig. 25. Brain A. Occipital conv. Strip *a* of Plate VIII, Fig. 24, magnified 100 diameters.

Plate IX. Fig. 26. Brain A. Large pyramidal cells from layer of large pyramidal cells of the hippocampus major or cornu ammonis. Section 6 2-3 microns in thickness, stained with methylene violet, and magnified 1,400 diameters.

Plate IX. Fig. 27. Brain A. Purkinje and granular cells from cortex

of left cerebellar hemisphere. External surface. Section 10 microns in thickness, stained with methylene blue, and magnified 1,400 diameters.

Plate X. Fig. 28. Brain A. Multipolar ganglion cell from ant. horn of lumbar enlargement of spinal cord. Section 6 2-3 microns in thickness, stained with methylene violet, and magnified 1,400 diameters.

Plate X. Fig. 29. Brain B. Left hemisphere. Case of dementia paralytica dying in advanced stage of terminal dementia with contractions. Normal size.

Plate X. Fig. 30. Brain B. First frontal conv. taken from point indicated by letter *K* in Plate X, Fig. 29. Section 10 microns in thickness, stained with methylene blue, and magnified 14 diameters.

Plate XI. Fig. 31. Brain B. First frontal conv. Strip *a* of section adjacent to that shown in Plate X, Fig. 30, 6 2-3 microns in thickness, stained with methylene blue, and magnified 100 diameters.

Plate XI. Fig. 32. Brain B. Ant. cent. conv. Upper third taken from point indicated by letter *D* in Plate X, Fig. 29. Section 10 microns in thickness, stained with methylene violet, and magnified 14 diameters.

Plate XI. Fig. 33. Brain B. Ant. cent. conv. Upper third. Strip *a* of section adjacent to that shown in Plate XI, Fig. 32. Section 6 2-3 microns in thickness, stained with methylene violet, and magnified 100 diameters.

Plate XI. Fig. 34. Brain A. Ant. cent. conv. Upper third taken from point indicated by letter *L* in Plate X, Fig. 29. Section 6 2-3 microns in thickness, stained with methylene violet, and magnified 14 diameters.

Plate XII. Fig. 35. Brain B. Ant. cent. conv. Upper third. Giant pyramidal cell *a* of Plate XI, Fig. 34, magnified 1,400 diameters.

Plate XII. Fig. 36. Brain B. Ant. cent. conv. Upper third. Giant pyramidal cell *b* of Plate XI, Fig. 34, magnified 1,400 diameters.

Plate XII. Fig. 37. Brain B. Ant. cent. conv. Upper third. Giant pyramidal cell from section adjacent to that shown in Plate XI, Fig. 34, magnified 1,400 diameters.

Plate XII. Fig. 38. Brain B. Ant. cent. conv. Upper third. Giant pyramidal cell from same section as that from which Plate XII, Fig. 37, was taken, magnified 1,400 diameters. Shows advanced chromatolysis.

Plate XIII. Fig. 39. Brain B. Ant. cent. conv. Upper third taken from point indicated by letter *O* in Plate X, Fig. 29. Section is 10 microns in thickness, stained with methylene violet, and magnified 15 diameters.

Plate XIII. Fig. 40. Brain B. Post. cent. conv. Middle third taken from region indicated by letter *B* in Plate X, Fig. 29. Section is 6 2-3 microns in thickness, stained with methylene blue, and magnified 14 diameters.

Plate XIII. Fig. 41. Brain B. Post. cent. conv. Middle third. Strip *a* of Plate XIII, Fig. 40, showing first and second layers of cortex and upper part of third layer, magnified 100 diameters.

Plate XIV. Fig. 42. Brain B. Supra-marginal conv. of parietal region taken from point indicated by letter *H* in Plate X, Fig. 29. Section 6 2-3 microns in thickness, stained with methylene violet, and magnified 14 diameters.

Plate XIV. Fig. 43. Brain B. Supra-marginal conv. of parietal region. Strip *a* of Plate XIV, Fig. 42, magnified 100 diameters.

Plate XIV. Fig. 44. Brain B. First temp. conv. taken from region indicated by letter *I* in Plate X, Fig. 29. Section 6 2-3 microns in thickness, stained with methylene violet, and magnified 14 diameters.

Plate XV. Fig. 45. Brain B. First temp. conv. Strip *a* of Plate XIV, Fig. 44, and magnified 100 diameters.

Plate XV, Fig. 46. Brain B. Occipital conv. taken from point indicated by letter *J* in Plate X, Fig. 29. Section 6 2-3 microns in thickness, stained with methylene violet and magnified 14 diameters.

Plate XV, Fig. 47. Brain B. Occipital conv. Strip *a* of Plate XV, Fig. 46, magnified 100 diameters.

ADDENDA.

The writer also wishes to acknowledge with thanks the appropriation of two hundred dollars from the Dyckman Fund of the Department of Zoölogy, Columbia University, towards the publication of this thesis.

Periscope.

ALLGEMEINE ZEITSCHRIFT FÜR PSYCHIATRIE

(Vol. 60, 1903, No. 3.)

1. Obituary. R. v. Krafft-Ebing. H. SCHULE.
2. Brain Weights in Dementia Paralytica. G. ILBERG.
3. Periodical Insanity. C. EISATH.
4. The Muscle Sense and Its Representation by Maupassant. O. KLUGE.
5. Contribution to Idiot Statistics. G. HEIMAN.
6. Case of Mania of Forensic Interest. KÖLPIN.
7. Psychiatric Therapy. P. PRENGOWSKY.
8. Statistical Study.

1. *Obituary, R. von Krafft-Ebing.*—A short sketch of the life and work of the late Prof. von Krafft-Ebing.

2. *Brain Weight in Dementia Paralytica.*—The author gives the results of his investigations as to the weight of the brain and its different parts in 102 male paretics dying in the Royal Saxon Asylum at Sonnenstein. His method was first to weigh the brain (removed after the thoracic organs), then dissecting it after Meynert's method, to weigh separately, the brain muscle, brain stem, and cerebellum; next dividing the brain mantle, to weigh right and left hemispheres, later the frontal, and temporo-parieto-occipital portions, and the basal ganglia of each hemisphere. His results are arranged according to the length of body of the patients in tabular form, being compared in the same table with the weights given Pflüger as normal for men of corresponding sizes. In other columns are placed the proportion of the weights of the different parts to the total brain weight, duration of the disease in months, and ages of the patients at death. His figures show that in long duration of the disease all parts of the brain lose weight, most of all the brain mantle and especially the frontal lobes, and that in the majority of cases there is a difference in weight between the two hemispheres, the right being more often the lighter. The article, a very comprehensive one, bristles with figures. It should be consulted in the original by those specially interested.

3. *Periodical Insanity.*—The prevailing views with regard to periodical insanity are those of Hitzig and Pilcz on the one hand, and of Kraepelin on the other. The former class as periodical, all mental disturbances occurring at regular intervals, each time with the same peculiar symptoms, so recognize a periodical mania, melancholia, amentia, paranoia, etc. Kraepelin on the contrary thinks that it is impossible to set a limit within which the periodical attacks begin and run their course, since irregularity is the rule, and regularity the exception, the latter being practically never found except in manio-melancholic insanity. Accordingly he does not admit that there is a periodical mania or periodical melancholia, but feels sure that sufficiently careful and long-continued observation will always detect a depressed and an exalted stage, though either may be but little marked.

As a contribution to the literature of the subject, Eisath gives careful clinical histories of five cases, each observed for a number of years. In the first case, that of a female beginning at twenty-five; during twenty-one years, there were 13 attacks marked by depression, never exaltation. In the second, that of a female beginning at forty-six, there were 20 attacks of exaltation in 16 years, never depression. The third case was that of a woman in whom between the ages of seventeen and sixty-seven years, there were 12 severe attacks occurring at irregular intervals, and showing mixed

excitement and depression, manio-melancholic insanity. The fourth and fifth cases respectively in a man and in a woman, though presenting a somewhat complicating symptom-complex, are regarded by the author as examples of dementia precox with a periodical course. Only in his second case does he think that the symptoms occurred with sufficient definiteness and regularity, to fulfil the requirements of Hitzig's and Pilcz's classification. On the whole he is inclined to take a position between that of Hitzig and Pilcz, and that of Kraepelin inclining to the opinion of the latter that periodicity alone is not a satisfactory criterion for the separation of psychical diseases.

4. *On the Muscle Sense and Its Representation by Maupassant.*—After a short discussion of the muscle sense, from which our notion of the Ego, or sense of self, is in his opinion, almost entirely derived, the author studies the manifestations of altered self feeling due to the pathological disturbance of this sense as presented by Maupassant, especially in his novel "La Horla," which was written after the gifted Frenchman was doubtless already in the first stage of general paresis.

5. *A Contribution to Idiot Statistics.*—Unsuitable for abstraction.

6. *A Case of Mania of Forensic Interest.*—Case of a man of twenty-six years, arrested for a number of small swindling operations, and found to be suffering from not very well marked mania. Of chief interest is the marked family and hereditary character of the mental trouble, which the author exposes at some length.

7. *Psychiatry Therapy.*—The author calls attention to the skin as an organ of elimination and thinks that a failure of this organ to perform its function may be at the bottom of some psychoses of possibly toxic origin. In a case diagnosed as "periodical neurasthenia" he claims to have been able by the local application of cold, to produce an array of symptoms affecting the skin, the circulatory organs, and sometimes the digestive apparatus, with mental manifestations corresponding closely to those of katatonia, all of which could be made to disappear, by warming up the skin again. From clinical study, and from the above experimental evidence, he urges the advisability of avoiding drugs which tend to produce arterial spasm, caffeine especially. The symptoms described he attributes to the production of arterial spasm, the consequent suppression of transpiration through the skin, and urges the rationality of encouraging sweating, and especially of the use of the prolonged warm bath as recommended by Kraepelin and others.

8. *Statistics of Patients in Institutions for the Insane, Idiotic and Epileptic.*—Tabular only. Unsuitable for abstraction.

C. L. ALLEN (Trenton).

MISCELLANY

PATHOGENY OF THE ACUTE PSYCHOSES. H. Berger (Berliner klinische Wochenschrift, July 27, 1903).

This is still one of the unsolved problems of psychiatry, and none of the numerous theories advanced have stood the test of experimental trials. The view which has lately met with the most support is that these phenomena depend on delicate chemical changes in the cortical cells, which occur without the production of any morphological alterations in the cells themselves. In this way the theory of a circulating toxin in the blood has gradually gained credence. Experimental proof of this theory has been sought by the author, who made the first trial upon himself. He injected, at intervals, serum, blood and cerebrospinal fluid from a patient suffering from acute dementia with hallucinations, without the least effect. This seems to show that the toxin, if present, must already be firmly united with the cerebral cells before the acute symptoms appear. In the belief that the toxin may

have been found during the prodromal stage, the author also injected subcutaneously blood from a patient who was developing symptoms during her puerperium, which later turned out to be a dementia precox. No effect was seen, but blood taken from the same patient four weeks later, during a fresh attack, and injected, was shortly followed by vertigo, and later by cardiac palpitation, cerebral pressure, and a marked feeling of fear. All these symptoms subsided on the following day. A similar experiment with the blood taken from a more advanced case in a condition of stupor at the time, was also followed by results which were much more marked and severe and did not subside for a week. The experiments were then continued on animals, and a basis secured for further investigation in regard to the changes which specific toxins contained in the circulating blood may cause in the central nervous system. The details are not suitable for a brief abstract. They consist mainly of observations made with the serum secured from the goat, which had been made neurotoxic for dogs by the continued subcutaneous injection of triturated cerebrum from the brains of dogs. Intracerebral injections of this goat serum in dogs was followed by well-marked pathological changes in the pyramidal cells of the cerebral cortex and later on a large aggregation of leucocytes around these degenerated cells. Similar pathological conditions have been found in patients afflicted with acute psychoses, and also in other cerebral diseases, but the author is not as yet prepared to draw final conclusions until further proof has been secured.

JELLIFFE.

MORPHINE AND OTHER PHENANTHRENE DERIVATIVES. P. Bergell and R. Pschorr (*Zeitschrift f. Physiologische Chemie*, Vol. 38, Nos. 1, 2).

It is taught by chemists that morphine is derived from phenanthrene, a cyclic hydrocarbon having the symbol $C_{14}H_{10}$; the basic character of morphine being determined, like that of all other alkaloids, by the presence of nitrogen. According to the authors, all the investigations that have so far sought to attribute the physiological action of morphine to single atomic groups, have been carried out almost entirely upon nitrogenous compounds related to morphine. The authors sought to determine to what extent the physiological action of morphine depends upon non-nitrogenous molecular groups, more particularly phenanthrene and its non-nitrogenous derivatives. They find that phenanthrene itself is inert, but that oxy-phenanthrene, or phenanthrol ($C_{14}H_9OH$) produces in mammals severe tetanic seizures. The position of the hydroxyl group in the molecule has no influence on the physiological effect. Similar symptom-complexes are produced by a carbon- and a sulpho-acid of phenanthrene. No narcotic effect was observed in this entire group. To what extent the effect of phenanthrol may be brought into relation to that of morphine which contains the phenanthrol-complex, and whether the behavior of this class of bodies affords any explanation of the tetanic components of morphine-action—both of these questions the authors consider as still unanswerable. Substantially different from the action of phenanthrol and its carbon-acids, is that of derivatives of phenanthrene-equinone. These substances, both in the test-tube and in the organism, show a decided power of forming methemoglobin, which power is to be attributed to the prepondering influence of the quinone group. None of these compounds produces the above described tetanic state. Epiosin, a methyl derivative of phenanthrene, also produces methemoglobinemia. It is significant that in pigeons, which show an extraordinary resistance toward morphine, severe toxic manifestations are produced by epiosin. Evidently there is some connection between the narcotic and other manifestations of morphine and the methemoglobinemia produced by some of its derivatives.

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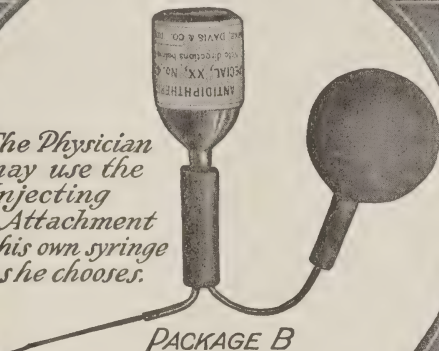
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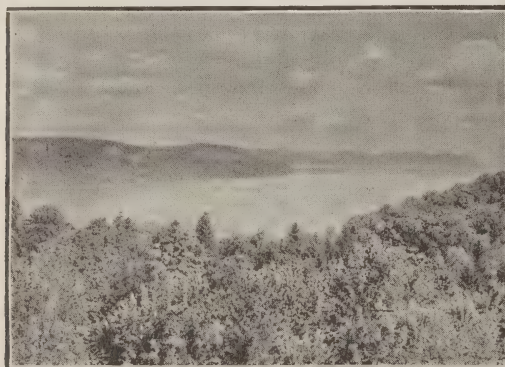
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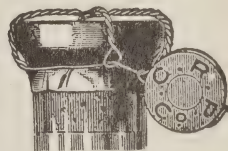
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The ease and facility with which the children of actors adapt themselves to the calling of their parents is rather strikingly illustrated in several conspicuous cases among the members of F. F. Proctor's big stock companies in his various theaters. Take pretty and clever Florence Reed, for example. The daughter of a famous comedian, she made her first footlight bow with scarcely any training, and yet, so naturally did acting come to her, that in less than two years she has gained the distinction of playing "comedy leads" in a Broadway theater—namely, Mr. Proctor's Fifth Avenue. But Miss Reed is not the only example of inherited histrionic ability in the Proctor ranks. There is that exceedingly capable young character actor, Albert Roberts, son of famous "Bob" Roberts, the comedian and stage manager; there is clever young Louis Owen, son of that fine old Shakespearean actor, William F. Owen, once one of Augustin Daly's best liked players; there is little Louis Bond, who, while thus far only seen in minor roles, gives frequent evidence that he has no little of the cleverness that for years has made a public favorite of his father, Frederic Bond, and finally, there is Gerald Griffin, the popular "character" man of the Proctor forces, who, although hardly a stripling, is not yet so old that he cannot point back with pride to his distinguished ancestor, Mack Griffin, the Irish tragedian, and the best Othello ever seen in Dublin.

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actors, including the stock companies, vaudeville performers, etc. In all, nearly 1,000 wage earners draw weekly stipends from the Proctor treasury.

The perennially graceful minstrel comedian, George H. Primrose, who made a fortune with his own shows, and then retired to enjoy a period of well-earned ease and competence, is the latest example of a man who simply can't "loaf." After six months of luxurious idleness, the popular blackface king has declared that he didn't feel that life was worth living unless he could get into burnt cork again. Besides, he has taken a fatherly interest in those diminutive dancers, the Foley Brothers, who assist him in his specialty, and for their sake also he has decided to return to the footlights. Primrose owns the largest private estate in Mt. Vernon, N. Y., a stable of fast trotters, a steam yacht, and plenty of government bonds. Yet he has deliberately chosen to keep on working, and his decision will be hailed with delight by all who have ever enjoyed his performance. Mr. Primrose is to appear in the F. F. Proctor theaters during the winter.

One of the greatest and quickest money-making plays ever presented on the American stage is to have a brilliant production at F. F. Proctor's stock theaters (the Fifth Avenue and the 125th Street) before snow flies. That play is "Trilby." When Paul Potter (who was once a clever newspaper reporter and a keen dramatic critic) made his dramatization of Du Maurier's famous story, he offered it in vain to at least a dozen New York managers. A. M. Palmer, then in control of Wallack's Theater, at last put the play on, but with hardly any hope of its success. Yet it opened to a packed house, made a profound, immediate and genuine hit, and incidentally made a rich man of leisure out of its author, who now lives on his extensive estate in Switzerland, the ideal life of a man of letters and of leisure.

LEUCORRHEA.

I find Aletris Cordial Rio to be an excellent and palatable preparation. I have used it in cases of dysmenorrhea, irritable ovary, uterine congestion, leucorrhea, and endometritis, with the best of results. In a case of irritable ovary that had resisted all treatment for four years, I prescribed Aletris Cordial Rio, and after taking for four days the pain was entirely relieved.

T. R. DICE, M.D., Utica, Mo.





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